STIC Search Report
Biotech-Chem Library

STIC Database Tracking Number: 130657

TO: John Pak **Location: 4a25 / 4c70** **Tuesday, August 31, 2004** **Art Unit: 1616** **Phone: 272-0620** **Serial Number: 10 / 070486**	**From: Jan Delaval** **Location: Biotech-Chem Library** **Rem 1A51** **Phone: 272-2504** **jan.delaval@uspto.gov**

Search Notes

130657

Scientific and Technical Information Center

Scientific & Technical Information Center USPTO

Request a Search

Search requests relating to **published applications, patent families, and litigation** may be submitted by filling out this form and clicking on "Send."

For all other search requests, fill out the form, print, and submit the printout with any attachments to the STIC facility serving your Technology Center.

Tech Center:

◉ TC 1600 ○ TC 1700 ○ TC 2100 ○ TC 2600 ○ TC 2800
○ TC2900 ○ TC 3600 ○ TC 3700 ○ Law Lib ○ Other

Enter your Contact Information below:

Name: John Pak

Employee Number: 69320 Phone: 571-272-0620

Art Unit or Office: 1616 Building & Room Number: REM 4A25

Mailbox REM 4C70

Enter the case serial number (Required): 10/070,486

If not related to a patent application, please enter NA here.

Class / Subclass(es)

Earliest Priority Filing Date: 9/13/1999

Jan 8/3/04

Format preferred for results:
☑ *Paper* ☐ *Diskette* ☐ *E-mail*

Provide detailed information on your search topic:

- In your own words, describe in detail the concepts or subjects you want us to search.
- Include synonyms, keywords, and acronyms. Define terms that have special meanings.
- *For Chemical Structure Searches Only*
  Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers
- *For Sequence Searches Only*
  Include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.
- *For Foreign Patent Family Searches Only*
  Include the country name and patent number.
- Provide examples or give us relevant citations, authors, etc., if known.
- FAX or send the **abstract, pertinent claims** (not all of the claims), **drawings, or chemical structures** to your EIC or branch library.

Enter your Search Topic Information below:

I. Method of treating sepsis by administering a composition wherein 1) lipid provides greater than 35% of the total energy of the composition,
2) ratio of n-6 fatty acid to n-3 fatty acid is between 2 and 7 (that is to say, n-6 is present at 2 to 7 times higher amount than n-3 fatty acids).

II. Same method as above, but with an additional feature: 25-70% of the lipids in the composition is from MCT (medium chain triglycerides) and there is less than 15% by weight saturated fatty acids, not counting the MCTs.

---

n-6 fatty acids (also known as omega-6 fatty acids): linoleic acid, gamma-linolenic acid, dihomo-gamma-linolenic acid, arachidonic acid, docosadienoic acid

n-3 fatty acids (also known as omega-3 fatty acids): EPA (eicosapentaenoic acid), DPA (docosapentaenoic acid), DHA (docosahexaenoic acid), ALA (alpha linolenic acid).

Special Instructions and Other Comments:
(For fastest service, let us know the best times to contact you, in case the searcher needs further clarification on your search.)

Press ALT + F, then P to print this screen for your own information.

SEND RESET

```
=> d his

     (FILE 'HOME' ENTERED AT 09:35:24 ON 31 AUG 2004)
               SET COST OFF

     FILE 'HCAPLUS' ENTERED AT 09:35:33 ON 31 AUG 2004
L1             1 S (WO2000-EP8731 OR EP99-118173)/AP,PRN
               E NESTLE/PA,CS
L2          2617 S NESTLE?/PA,CS
               E TURINI M/AU
L3            25 S E3-E6
               E ROESSLE C/AU
L4             8 S E3,E4
               E ROSSLE C/AU
L5             7 S E3,E4
               E BREUILLE D/AU
L6            34 S E3,E4
               E CROZIER W/AU
L7             5 S E3,E9,E10
               E WILLI G/AU
               E CROZIER G/AU
L8            24 S E3-E8
               E FINOT P/AU
L9            80 S E4-E7
               E RICHELLE M/AU
L10           45 S E3,E4,E8
               E DUTOT G/AU
L11           11 S E3,E5

     FILE 'REGISTRY' ENTERED AT 09:45:51 ON 31 AUG 2004
L12            6 S (LINOLEIC ACID OR Γ-LINOLENIC ACID OR DIHOMO-Γ-LI
L13            9 S (EICOSAPENTAENOIC ACID OR DOCOSAPENTAENOIC ACID OR DOCOSAHEXA
L14            1 S 92661-11-5
L15            7 S L12,L14
L16            1 S DIHOMO-Γ-LINOLEINIC ACID/CN
L17            7 S L15,L16

     FILE 'HCAPLUS' ENTERED AT 09:52:10 ON 31 AUG 2004
L18        52030 S L17
L19         1471 S L17(L) FFD/RL
L20         1280 S L17(L) THU/RL
L21        20593 S L18 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L22        63109 S (ARACHIDONIC OR LINOLEIC OR GAMMA LINOLENIC OR DIHOMO GAMMA()
L23        24880 S L22 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
               E FATTY ACIDS/CT
               E FATTY ACIDS (L) N/CT
               E FATTY ACIDS (L) OMEGA/CT
               E FATTY ACIDS (L) POLYUNSAT/CT
L24         2403 S E13,E16
L25         2519 S FATTY ACID?/CT (L) (N6 OR (N OR OMEGA)()6 OR OMEGA6)
L26          276 S L24,L25 (L) FFD/RL
L27          168 S L24,L25 (L) THU/RL
L28         2167 S L24,L25 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?
L29        30232 S L19,L20,L21,L23,L26,L27,L28
L30        26049 S L13
L31         1830 S L13 (L) FFD/RL
L32         1316 S L13 (L) THU/RL
L33        14009 S L30 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L34        12812 S (EICOSAPENTAENOIC OR DOCOSAPENTAENOIC OR DOCOSAHEXAENOIC OR A
L35         8135 S L34 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
               E FATTY ACIDS (L) POLYUNSAT/CT
L36         5489 S E12,E14,E15
L37         5703 S FATTY ACIDS?/CT (L) (N3 OR (N OR OMEGA)()3 OR OMEGA3)
```

```
L38          855 S L36,L37 (L) FFD/RL
L39          654 S L36,L37 (L) THU/RL
L40         5131 S L36,L37 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?
L41        17717 S L31,L32,L33,L35,L38,L39,L40
L42        11526 S L29 AND L41
L43        21471 S L18-L29 AND L30-L41
L44         5431 S L43 AND LIPID#/CW
                 E MEDIUM CHAIN TRIGLYCERIDE/CT
                 E E11+ALL
L45         1538 S E2
L46         2489 S ((MED OR MEDIUM OR M)()CHAIN)(L)(GLYCERIDE OR TRIGLYCERIDE)
                 E FISH OIL/CT
                 E E5+ALL
L47         4257 S E2
L48         1570 S L47 (L) (FFD/RL OR THU/RL)
L49         3744 S L47 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L50          715 S L47-L49 AND LIPID#/CW
L51           85 S L47-L49 AND L45,L46
L52         5950 S L44,L50,L51
                 E SEPSIS/CT
L53         7317 S E3,E5-E10
                 E E8+ALL
L54         8136 S E1,E2,E3
                 E E3+ALL
L55         3750 S E4+NT
                 E E3+ALL
L56        10652 S E3+NT
                 E SHOCK/CT
L57         4729 S E4-E12
                 E E4+ALL
L58        13891 S E8,E9,E7+NT
                 E E26+ALL
L59         6857 S E3,E2+NT
L60            8 S L52 AND L53-L59
L61          152 S L1-L11 AND L18-L60
L62           17 S L61 AND L47-L49
L63           55 S L61 AND L42,L43
L64           30 S L63 AND LIPID
L65           41 S L62,L64
L66           23 S L62,L63 NOT L65
L67            8 S L52 AND ?SEPSI?
L68           13 S L52 AND ?SEPTI?
L69           17 S L60,L67,L68
L70            1 S L61 AND L69
L71           14 S L69 AND (PY<=1999 OR PRY<=1999 OR AY<=1999)
L72            3 S L69 NOT L70,L71
L73            1 S L72 AND SEPTIC RAT
                 SEL DN AN L71 2 6 12 13
L74           10 S L71 NOT E1-E12
L75           11 S L73,L74,L70
L76           11 S L75 AND (N6 OR N 6 OR OMEGA6 OR OMEGA 6 OR N3 OR N 3 OR OMEGA
L77            8 S L76 AND (LINOLEIC OR ?LINOLENIC? OR ARACHINDONIC OR DOCOSADIE
L78           11 S L75-L77 AND L1-L11,L18-L77

=> fil hcaplus
FILE 'HCAPLUS' ENTERED AT 10:26:26 ON 31 AUG 2004
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)
```

FILE COVERS 1907 - 31 Aug 2004 VOL 141 ISS 10
FILE LAST UPDATED: 30 Aug 2004 (20040830/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all hitstr tot l78

L78 ANSWER 1 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:208096 HCAPLUS
DN 134:236858
ED Entered STN: 22 Mar 2001
TI High **lipid** diet for prevention or treatment of **sepsis** or inflammatory shock
IN **Turini, Marco; Roessle, Claudia; Breuille, Denis; Crozier-Willi, Gayle; Finot, Paul-Andre; Richelle, Myriam; Dutot, Guy**
PA Societe des Produits **Nestle** S.A., Switz.
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-00
CC 18-5 (Animal **Nutrition**)
Section cross-reference(s): **1**, **17**, **63**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001019356	A2	20010322	WO 2000-EP8731	20000907 <--
	WO 2001019356	A3	20010517		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1090636	A1	20010411	EP 1999-118173	19990913 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	AU 2000068430	A5	20010417	AU 2000-68430	20000907 <--
	BR 2000013958	A	20020514	BR 2000-13958	20000907 <--
	EP 1216041	A2	20020626	EP 2000-956522	20000907 <--
	EP 1216041	B1	20040204		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
	AT 258792	E	20040215	AT 2000-956522	20000907 <--
PRAI	EP 1999-118173	A	19990913 <--		
	WO 2000-EP8731	W	20000907 <--		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES

WO 2001019356 ICM A61K031-00

AB A composition for use as a medicament, functional food, or nutritional product is described which comprises at least one **lipid** which provides > 35% total energy of the composition A preferred embodiment comprises an **n-6/n-3 fatty acid** ratio of about 2:1 to 7:1. In addition, a method of preparing the composition, use of the composition in the manufacture of a medicament or nutritional product, and a method of treatment or prevention of **sepsis** or inflammatory shock comprising administering an effective amount of the composition are described. An example showing that a high **lipid** diet (15% and 35% **lipids**) limits body weight loss in a rat model of **sepsis** was presented. A high-**lipid** diet had a beneficial effect for limitation of N loss induced by **sepsis**, suggesting a potential decrease of muscle proteolysis (which is dramatically increased in acute inflammatory conditions). It was particularly effective if the diet has been enriched with **lipids** before infection.

ST **lipid** omega **fatty acid** food nutrient; **sepsis** inflammatory shock **lipid** diet

IT Anti-inflammatory agents
Drug delivery systems
Food
Nutrients
Sepsis
(compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT Canola oil
Fatty acids, biological studies
Olive oil
Safflower oil
Soybean oil
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fats and Glyceridic oils, biological studies**
RL: **FFD (Food or feed use)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**fish**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Lipids**, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Diet**
(high-**lipid**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Shock (circulatory collapse)**
(inflammatory; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Glycerides, biological studies**
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**medium-chain**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT Fats and **Glyceridic** oils, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(milk; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fatty acids**, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**monounsatd.**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fatty acids, biological studies**
RL: **FFD (Food or feed use)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**polyunsatd.**, **n-3**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fatty acids, biological studies**
RL: **FFD (Food or feed use)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**polyunsatd.**, **omega-6**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fatty acids**, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**polyunsatd.**; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

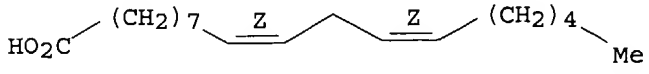
IT **Fatty acids**, biological studies
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(saturated; compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies **60-33-3**, **Linoleic acid**, biological studies 112-80-1, 9-Octadecenoic acid (9Z)-, biological studies **463-40-1**, α **-Linolenic acid** **506-26-3**, γ **-Linolenic acid** **506-32-1**, **Arachidonic acid** 544-63-8, Tetradecanoic acid, biological studies **6217-54-5**, DHA **10417-94-4**, **Eicosapentaenoic acid** **32839-34-2**, **Docosapentaenoic acid** **92661-11-5**, **Dihomo-γ -linoleinic acid**
RL: **FFD (Food or feed use)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **60-33-3**, **Linoleic acid**, biological studies **463-40-1**, α **-Linolenic acid** **506-26-3**, γ **-Linolenic acid** **506-32-1**, **Arachidonic acid** **6217-54-5**, DHA **10417-94-4**, **Eicosapentaenoic acid** **32839-34-2**, **Docosapentaenoic acid** **92661-11-5**, **Dihomo-γ -linoleinic acid**
RL: **FFD (Food or feed use)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(compns. of high-**lipid** diet for prevention or treatment of **sepsis** or inflammatory shock)

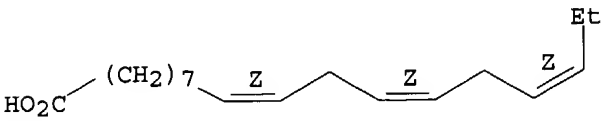
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



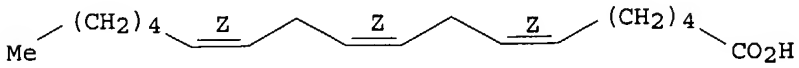
RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



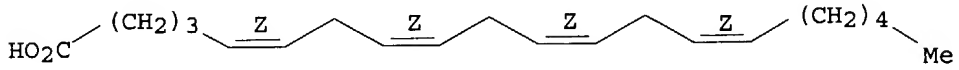
RN 506-26-3 HCAPLUS
CN 6,9,12-Octadecatrienoic acid, (6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



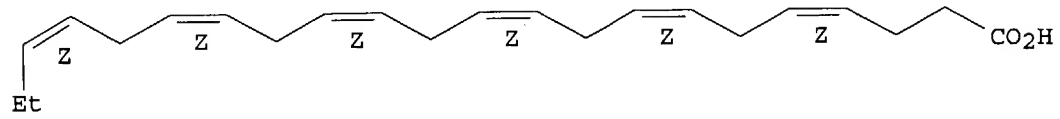
RN 506-32-1 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



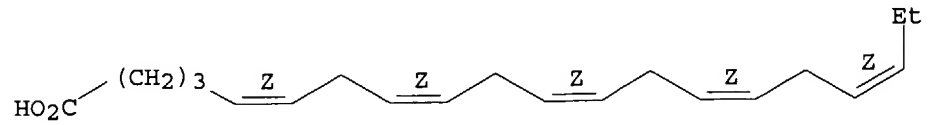
RN 6217-54-5 HCAPLUS
CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 32839-34-2 HCAPLUS
CN Docosapentaenoic acid, (Z,Z,Z,Z,Z)- (9CI) (CA INDEX NAME)

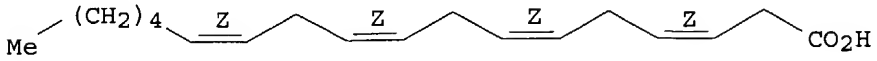
CM 1

CRN 112-85-6
CMF C22 H44 O2

$HO_2C-(CH_2)_{20}-Me$

RN 92661-11-5 HCAPLUS
CN 3,6,9,12-Octadecatetraenoic acid, (3Z,6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:237890 HCAPLUS
DN 133:104372
ED Entered STN: 13 Apr 2000
TI Effects of parenteral infusion with fish-oil or safflower-oil emulsion on hepatic **lipids**, plasma amino acids, and inflammatory mediators in **septic rats**
AU Chao, C. Y.; Yeh, S. L.; Lin, M. T.; Chen, W. J.
CS Institute of Nutrition and Health Science, Taipei Medical College, Taipei, Taiwan
SO Nutrition (New York) (2000), 16(4), 284-288
CODEN: NUTRER; ISSN: 0899-9007
PB Elsevier Science Inc.
DT Journal
LA English
CC 18-5 (Animal **Nutrition**)
AB This study was designed to investigate the effects of preinfusion with total parenteral nutrition (TPN) using fish-oil (FO) vs. safflower-oil (SO) emulsion as fat sources on hepatic **lipids**, plasma amino-acid profiles, and inflammatory-related mediators in **septic rats.** Normal rats, with internal jugular catheters, were assigned to two different groups and received TPN. TPN provided 300 kcal · kg-1 · d-1, with 40% of the non-protein energy as fat. All TPN solns. were isonitrogenous and identical in nutrient composition except for the fat emulsion, which was made of SO or FO. After receiving TPN for 6 d, each group of rats was further divided into control and **sepsis** subgroups. **Sepsis** was induced by cecal ligation and puncture; control rats received sham operation. All rats were classified into four groups as follows: FO control group (FOC; n = 7), FO **sepsis** group (FOS; n = 8), SO control group (SOC; n = 8), and SO **sepsis** group (SOS; n = 9). The results of the study demonstrated that plasma concns. of triacylglycerol and non-esterified **fatty acids** did not differ between the FO and SO groups, regardless of whether the animals were **septic.** SOS had significantly higher total **lipids** and cholesterol content in the liver than did the SOC group. The FOS group, however, showed no difference from the FOC group. Plasma leucine and isoleucine levels were significantly lower in the SOS group than in the SOC group, whereas no difference in these two amino acids was observed between the FOC and FOS groups. Plasma arginine levels were significantly lower in both **septic** groups than in the groups without **sepsis** when either FO or SO was infused. Plasma glutamine levels, however, did not differ across groups. No differences in interleukin-1β, interleukin-6, tumor necrosis factor-α, or leukotriene B4 concns. in peritoneal lavage fluid were observed between the two **septic** groups. These results suggest that catabolic reaction in **septic rats** preinfused with FO is not as obvious as those preinfused with SO. Compared with SO emulsion, TPN with FO emulsion prevents liver fat accumulation associated with

sepsis. However, parenterally administered FO had no beneficial effect in lowering cytokines and LTB4 levels in peritoneal lavage fluid in **septic rats** induced by cecal ligation and puncture.

ST fish oil liver **lipid** amino acid; safflower oil liver **lipid** amino acid; inflammation cytokines immunity fish safflower oil

IT **Fats and Glyceridic oils, biological studies**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**fish**; parenteral infusion of **fish**-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT Blood plasma
Inflammation
Liver
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT Safflower oil
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT Amino acids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT Cytokines
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT **Glycerides**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT Interleukins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT **Lipids**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT **Nutrition**, animal
(parenteral; parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

IT 56-86-0, Glutamic acid, biological studies 57-88-5, Cholesterol, biological studies 61-90-5, Leucine, biological studies 73-32-5, Isoleucine, biological studies 74-79-3, Arginine, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Association of Official Analytical Chemists; Official methods of analysis, 14th ed 1984, P276
(2) Bagby, G; Am J Physiol 1980, V238, PH325 HCAPLUS
(3) Bagby, G; Am J Physiol 1987, V253, PE59 HCAPLUS
(4) Bagby, G; Proc Soc Exp Biol Med 1981, V168, P395 HCAPLUS
(5) Baigrie, R; Br J Surg 1992, V79, P757 MEDLINE
(6) Bergstrom, J; J Appl Physiol 1974, V36, P693 MEDLINE
(7) Breil, I; Crit Care Med 1996, V24, P1893 MEDLINE
(8) Carlson, S; Clin Chim Acta 1977, V79, P575 HCAPLUS
(9) Chen, W; Clin Nutr 1996, V15, P24 HCAPLUS
(10) Clowes, G; Ann Surg 1974, V179, P684
(11) Dyerberg, J; Lancet 1978, V2, P117 HCAPLUS
(12) Ertel, W; Ann Surg 1991, V214, P141 MEDLINE
(13) Evoy, D; Nutrition 1998, V14, P612
(14) Folch, J; J Biol Chem 1957, V226, P497 MEDLINE
(15) Fong, Y; Surg Gynecol Obstet 1990, V170, P363 HCAPLUS
(16) Freund, H; Surgery 1978, V83, P611 HCAPLUS
(17) Gianotti, L; Ann Surg 1993, V217, P644 MEDLINE
(18) Grimminger, F; Clin Invest 1993, V71, P634 MEDLINE
(19) Grimminger, F; Eur J Clin Invest 1993, V23, P706 MEDLINE
(20) Halminski, M; J Nutr 1991, V121, P1554 HCAPLUS
(21) Harris, W; J Lipid Res 1989, V30, P785 HCAPLUS
(22) Herzberg, G; FASEB J 1988, V2, PA852
(23) Iriyama, K; JPEN 1988, V12, P60 MEDLINE
(24) Kromann, N; Acta Med Scand 1980, V208, P401 MEDLINE
(25) Lanza-Jacoby, S; Am J Physiol 1990, V258, PE678 HCAPLUS
(26) Lanza-Jacoby, S; JPEN 1992, V16, P353 MEDLINE
(27) Lanza-Jacoby, S; Lipids 1995, V30, P707 HCAPLUS
(28) Lowry, O; J Biol Chem 1951, V193, P265 HCAPLUS
(29) Morlion, B; Metabolism 1996, V45, P1208 HCAPLUS
(30) Odessey, R; Am J Physiol 1972, V223, P1376 HCAPLUS
(31) Odessey, R; J Biol Chem 1974, V249, P7623 HCAPLUS
(32) O'Donnell, T; Surgery 1976, V80, P192
(33) Rosato, E; Surg Forum 1990, V40, P25
(34) Rustan, A; Biochem J 1992, V283, P333 HCAPLUS
(35) Scholl, R; J Surg Res 1984, V37, P394 HCAPLUS
(36) Smith, R; J Liq Chromatogr 1985, V8, P1783 HCAPLUS
(37) Soloni, F; Clin Chem 1971, V17, P529 HCAPLUS
(38) Wichterman, K; J Surg Res 1980, V29, P189 MEDLINE
(39) Wilmore, D; J Am Coll Nutr 1985, V2, P3
(40) Yu, Y; Ann Surg 1988, V207, P421 MEDLINE
(41) Zanetti, G; J Immunol 1992, V148, P1890 HCAPLUS

L78 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:258015 HCAPLUS
DN 129:52707
ED Entered STN: 07 May 1998
TI **Lipid** mediators in inflammatory disorders
AU Heller, Axel; Koch, Thea; Schmeck, Joachim; Van Ackern, Klaus
CS Department of Anaesthesiology and Intensive Care Medicine, University of Dresden, Dresden, Germany
SO Drugs (**1998**), 55(4), 487-496
CODEN: DRUGAY; ISSN: 0012-6667
PB Adis International Ltd.
DT Journal; General Review
LA English
CC 14-0 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 2

AB A review, with 46 refs. During the past few decades, intensive collaborative research in the fields of chronic and acute inflammatory disorders has resulted in a better understanding of the pathophysiol. and diagnosis of these diseases. Modern therapeutic approaches are still not satisfactory and shock, **sepsis** and multiple organ failure remain the great challenge in intensive care medicine. However, the treatment of inflammatory diseases like rheumatoid arthritis, ulcerative colitis or psoriasis also represents an unresolved problem. Many factors contribute to the complex course of inflammatory reactions. Microbiol., immunol. and toxic agents can initiate the inflammatory response by activating a variety of humoral and cellular mediators. In the early phase of inflammation, excessive amts. of interleukins and **lipid** -mediators are released and play a crucial role in the pathogenesis of organ dysfunction. **Arachidonic acid** (AA), the mother substance of the pro-inflammatory eicosanoids, is released from membrane **phospholipids** in the course of inflammatory activation and is metabolized to prostaglandins and leukotrienes. Various strategies have been evaluated to control the excessive production of **lipid** mediators on different levels of biochem. pathways, such as inhibition of phospholipase A2, the trigger enzyme for release of AA, blockade of cyclo-oxygenase and lipoxygenase pathways and the development of receptor antagonists against platelet activating factor and leukotrienes. Some of these agents exert protective effects in different inflammatory disorders such as **septic** organ failure, rheumatoid arthritis or asthma, whereas others fail to do so. Encouraging results have been obtained by dietary supplementation with long chain ω -3 **fatty acids** like **eicosapentaenoic acid** (EPA). In states of inflammation, EPA is released to compete with AA for enzymic metabolism inducing the production of less inflammatory and chemotactic derivs.

ST **lipid** mediator inflammatory disease review

IT Disease, animal
Inflammation
(**lipid** mediators in inflammatory disorders)

IT Cytokines
Eicosanoids
Lipids, biological studies
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**lipid** mediators in inflammatory disorders)

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Allison, M; N Engl J Med 1992, V372, P749
(2) Arni, R; Toxicon 1996, V34, P827 HCAPLUS
(3) Bernhard, G; N Engl J Med 1997, V336, P912
(4) Breil, I; Crit Care Med 1996, V24, P1893 MEDLINE
(5) Christmann, B; Am Rev Respir Dis 1990, V142, P1272
(6) Dhainaut, J; Crit Care Med 1994, V22, P1720 MEDLINE
(7) Dyerberg, J; Am J Clin Nutr 1975, V28, P958 HCAPLUS
(8) Elbers, M; Clin Exp Dermatol 1994, V19, P453 MEDLINE
(9) Etienne, A; Agents Actions 1985, V17, P368 HCAPLUS
(10) Evans, D; Am J Respir Crit Care Med 1997, V156, P11 MEDLINE
(11) Evans, D; Thorax 1996, V51, P1178 MEDLINE
(12) Fischer, S; Nature 1984, V307, P165 HCAPLUS
(13) Froon, A; Shock 1996, V5, P313 MEDLINE
(14) Furst, D; Semin Arthritis Rheum 1997, V26(Suppl 1), P21
(15) Glaser, K; Skin Pharmacol 1995, V8, P300 HCAPLUS
(16) Goldman, D; Biochem Biophys Res Commun 1983, V117, P282 HCAPLUS
(17) Guth, B; Basic Res Cardiol 1997, V92, P181 HCAPLUS
(18) Harris, R; Int J Immunopharmacol 1995, V17, P147 HCAPLUS
(19) Heller, A; Anaesth Intensivmedizin 1996, V10, P517
(20) Hilliquin, P; J Rheumatol 1995, V22, P1651 HCAPLUS

(21) Hosford, D; J Crit Care 1990, V5, P115 HCAPLUS
(22) Hozawa, S; Am J Respir Crit Care Med 1995, V152, P1198 MEDLINE
(23) Israel, E; Am Rev Respir Dis 1993, V148, P1447 MEDLINE
(24) Jackson, D; Meeting report, Inflamm Res 1997, V46, P242
(25) Kingsnorth, A; Br J Surg 1995, V82, P1414 MEDLINE
(26) Koike, K; Surgery 1992, V112, P173 MEDLINE
(27) Kramer, R; Agents Actions Suppl 1995, V46, P65 HCAPLUS
(28) Laird, J; Inflamm Res 1997, V46, P203 MEDLINE
(29) Lee, T; Clin Sci 1988, V74, P467 HCAPLUS
(30) Lewis, R; Health effects of polyunsaturated fatty acids in seafoods 1986, P227
(31) Lewis, R; Proc Natl Acad Sci USA 1980, V77, P3710 HCAPLUS
(32) Marder, P; Br J Clin Pharmacol 1996, V42, P457 HCAPLUS
(33) Metz, C; J Crit Care 1990, V5, P206 HCAPLUS
(34) Miguelez, R; J Lipid Mediat Cell Signal 1996, V13, P35 HCAPLUS
(35) Neuhof, H; First Vienna Shock Forum, Pt A: pathophysiological role of mediators and mediator inhibitors in shock 1987, P289 MEDLINE
(36) Nickerson-Nutter, C; Arthritis Rheum 1996, V39, P515 HCAPLUS
(37) Rocha, F; J Lipid Mediat Cell Signal 1997, V16, P1 HCAPLUS
(38) Rola-Pleszczynski, M; J Lipid Med 1993, V6, P175 HCAPLUS
(39) Scott, K; Prostaglandin Relat Compounds 1992, PA151
(40) Seed, M; Inflamm Res 1997, V46, P279 MEDLINE
(41) Sheng, G; Gastroenterology 1997, V113(6), P1883 HCAPLUS
(42) Sperling, R; J Immunol 1987, V139, P4186 HCAPLUS
(43) Taylor, I; Lancet 1991, V337, P690 MEDLINE
(44) Weber, P; Biochem Soc Trans 1990, V18, P1045 HCAPLUS
(45) Yedgar, S; Agents Actions Suppl 1995, V46, P77 HCAPLUS
(46) Zwissler, B; Am J Respir Crit Care Med 1996, V154, P1671 MEDLINE

L78 ANSWER 4 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:505727 HCAPLUS
DN 127:126653
ED Entered STN: 11 Aug 1997
TI Pharmaceutical composition comprising L-carnitine or an alkanoyl L-carnitine in combination with a **polyunsaturated fatty acid** of the **omega-3** series for the prevention and the treatment of **lipid** metabolism disorders
IN Calvani, Menotti; Cavazza, Claudio
PA Sigma-Tau Industrie Farmaceutiche Riunite S.P.A., Italy
SO Eur. Pat. Appl., 7 pp.
CODEN: EPXXDW
DT Patent
LA English
IC ICM A61K031-205
ICI A61K031-205, A61K031-20
CC 63-6 (**Pharmaceuticals**)
Section cross-reference(s): **1**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 780124	A1	19970625	EP 1996-118681	19961121 <--
	EP 780124	B1	20011219		
	R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	AT 210977	E	20020115	AT 1996-118681	19961121 <--
	PT 780124	T	20020628	PT 1996-118681	19961121 <--
	ES 2169197	T3	20020701	ES 1996-118681	19961121 <--
	US 5753703	A	19980519	US 1996-755310	19961122 <--
	TW 522013	B	20030301	TW 1996-85114597	19961126 <--
	CA 2191645	AA	19970622	CA 1996-2191645	19961129 <--
	JP 09176005	A2	19970708	JP 1996-335456	19961216 <--
	ZA 9610769	A	19970709	ZA 1996-10769	19961220 <--
PRAI	IT 1995-RM835	A	19951221 <--		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
EP 780124	ICM	A61K031-205
	ICI	A61K031-205, A61K031-20
US 5753703	ECLA	A61K031/205; A61K031/22F <--

AB A novel therapeutic use of L-carnitine (I) or alkanoyl L-carnitine is disclosed in combination with a **polyunsatd. fatty acid** of the **omega-3** series, for the prevention and the treatment of **lipid** metabolism disorders and cardiovascular disorders. Rats with exptl. induced atherosclerotic lesions were treated with 100 mg I/kg and 2 mL fish oil/kg (containing 18% **eicosapentaenoic acid** and 12% **docosahexaenoic acid**) for 6 wk. No lesions were detectable in the treated group, revealing a surprising degree of synergism between I and **polyunsatd. fatty acids** of the **omega-3** series. A pharmaceutical composition contained I 250, **polyunsatd. fatty acids** of fish 1 (containing 350 mg **eicosapentaenoic acid** and 150 mg **docosahexaenoic acid**), and α-tocopherol acetate 1 mg.

ST pharmaceutical carnitine **polyunsatd** omega **fatty acid**; **lipid** metab disorder omega **fatty acid**; fish oil **eicosapentaenoic acid** antiatherosclerotic; **docosahexaenoic acid** fish oil antiatherosclerotic

IT Antiarteriosclerotics
(antiatherosclerotics; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(capsules; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **Fats and Glyceridic oils, biological studies**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**fish**; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(granules; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(liposomes; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(liqs.; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **Lipids**, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(metabolism, disorders; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention

and treatment of **lipid** metabolism disorders)

IT Salts, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mineral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Nerve, disease
(neuropathy, diabetic, peripheral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(oral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(parenterals; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Nerve, disease
(peripheral, diabetic neuropathy; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Blood vessel, disease
(peripheral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **Anaphylaxis**
Anti-inflammatory agents
Anticoagulants
Antioxidants
Cardiovascular agents
Drug delivery systems
Hypertriglyceridemia
Propolis
Shock (circulatory collapse)
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Corn oil
Vitamins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **Fatty acids, biological studies**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**polyunsatd., n-3**; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty**

acid for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(powders; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(rectal; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **Shock (circulatory collapse)**
(**septic**; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT Drug delivery systems
(solids; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

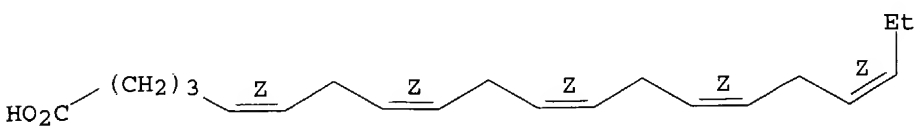
IT Drug delivery systems
(tablets; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT 112-85-6, Behenic acid 541-15-1, L-Carnitine 3040-38-8, Acetyl L-carnitine **10417-94-4**, **Eicosapentaenoic acid** 20064-19-1, Propionyl L-carnitine 25576-40-3, Butyryl L-carnitine 31023-24-2, Isovaleryl L-carnitine 40225-14-7, Valeryl L-carnitine 81926-94-5 86227-47-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

IT **10417-94-4**, **Eicosapentaenoic acid**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)

RN 10417-94-4 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 5 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:494170 HCAPLUS
DN 125:132809

ED Entered STN: 20 Aug 1996
TI Bioactive agent-containing biocomplex for correcting biological information transfer using three biological information blocks
IN Danielov, Michael M.
PA Dns Scientific, Inc., USA
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K038-21
ICS A61K039-395; A61K031-55; A61K031-44; A61K031-24
CC 1-12 (**Pharmacology**)
Section cross-reference(s): 2, 62, **63**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9617621	A1	19960613	WO 1995-US15919	19951206 <--
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	US 5885974	A	19990323	US 1994-350234	19941206 <--
	AU 9645108	A1	19960626	AU 1996-45108	19951206 <--
	US 6303588	B1	20011016	US 1999-228384	19990112 <--
PRAI	US 1994-350234	A	19941206 <--		
	WO 1995-US15919	W	19951206 <--		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9617621	ICM	A61K038-21
	ICS	A61K039-395; A61K031-55; A61K031-44; A61K031-24
WO 9617621	ECLA	A61K007/06C4F; A61K008/60C; A61K008/63; A61K008/64; A61K008/67; A61K008/67F; A61K008/67F3; A61K008/67L; A61K038/22; A61K038/33; A61K038/35; A61K038/39; A61K038/45; A61K038/46; A61K038/4; A61K045/06; A61K045/06; A61Q019/00; A61K007/48C4F; A61K007/48C18; A61K008/06C; A61K008/365; A61K; A61K008/55C <--
US 5885974	ECLA	A61K007/06C4F; A61K007/06C18; A61K007/06C16M; A61K007/06C20; A61K007/48C4F; A61K007/48C18; A61K007/48C16D; A61K007/48C22; A61K009/127M; A61K038/22; A61K038/33; A61K038/35; A61K038/39; A61K038/45; A61K008/46; A61K038/48; A61K045/06; A61K045/06 <--

AB Methods are disclosed for correcting biol. information transfer in a patient in need of such therapy which comprise administration of a composition comprising a therapeutically effective amount of a biocomplex comprising ≥1 bioactive agent from each of the 3 informational blocks of biol. information transfer, each agent present in an amount sufficient to correct the biol. information transfer of the patient under treatment and resulting in the resumption of normal cell metabolism, and the amount being less than the buffering amount of said agent; together with a carrier therefor.

ST biol information transfer block therapeutic; cell metab information transfer biocomplex therapeutic

IT Acne
Alopecia
Animal cell
Antioxidants
Circulation
Cosmetics

Eczema
Metabolism
Pharmaceutical dosage forms
Pharmaceuticals
Pruritus
Psoriasis
Seborrhea
Signal transduction, biological
Skin, disease
Therapeutics
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Albumins, biological studies
Calmodulins
Carbohydrates and Sugars, biological studies
Catecholamines
Cerebrosides
Coenzymes
Collagens, biological studies
Elastins
Gelatins, biological studies
Glycolipids
Lipids, biological studies
Orosomucoids
Peptides, biological studies
Phosphatidic acids
Phosphatidylcholines, biological studies
Phosphatidylethanolamines
Phosphatidylinositols
Phosphatidylserines
Phosphoinositides
Phospholipids, biological studies
Prostaglandins
Protamines
Proteins, biological studies
Sphingolipids
Steroids, biological studies
Sulfatides
Vitamins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Animal growth regulator receptors
Estrogen receptors
Prostaglandin receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Brain
(extract; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Shock**
(post-trauma; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Cell membrane
(substitute cell membrane delivery system; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Prostaglandins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

(Biological study); PROC (Process)
(A, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Prostaglandins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(D, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Prostaglandins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(E, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(animal growth regulator, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Skin
(cellulite, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Glycerides**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(di-, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Phosphoinositides
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(di-, 4-phosphates, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Skin, disease
(dry, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(estrogen, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Corticosteroid receptors
Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(glucocorticosteroid, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Lipoproteins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(high-d., bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Phosphatidylcholines, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hydrogenated, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Elastins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic

use); BIOL (Biological study); USES (Uses)
(hydrolyzates, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Lipoproteins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(low-d., bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Corticosteroid receptors
Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(mineralocorticosteroid, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Dermatitis
(neuro-, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Skin, disease
(oily, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Pharmaceutical** dosage forms
(ointments, creams, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Pharmaceutical** dosage forms
(ophthalmic, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Pharmaceutical** dosage forms
(parenterals, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(prostaglandin, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Sunburn and Suntan
(suntanning agents, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Pharmaceutical** dosage forms
(topical, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Injury
(trauma, shock following; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Phosphoinositides
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(tri-, 4,5-bis(phosphates), bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Collagens, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(type I, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Collagens, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(type II, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Collagens, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(type III, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Lipoproteins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(very-low-d., bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Skin, disease
(wrinkle, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(α2-adrenergic, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(β2-adrenergic, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT 60-92-4, Cyclic AMP
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT 50-14-6, Ergocalciferol 50-23-7, Hydrocortisone 50-28-2, β-Estradiol, biological studies 50-81-7, L-Ascorbic acid, biological studies 51-61-6, Dopamine, biological studies 52-39-1, Aldosterone 52-89-1, L-Cysteine hydrochloride 53-59-8, β-NADP 53-84-9, β-NAD 54-47-7, Pyridoxal-5-phosphate 55-31-2, Epinephrine hydrochloride 56-65-5, Adenosine triphosphate, biological studies 56-81-5D, 1,2,3-Propanetriol, 1,2-diacyl derivs. 56-89-3, L-Cystine, biological studies 57-11-4, Octadecanoic acid, biological studies 57-83-0, Progesterone, biological studies 57-87-4, Ergosterol 57-88-5, Cholesterol, biological studies 58-56-0, Pyridoxine hydrochloride 58-85-5, Biotin 58-95-7, α-Tocopherol acetate 59-30-3, Folic acid, biological studies 60-18-4, L-Tyrosine, biological studies **60-33-3**, 9,12-Octadecadienoic acid (Z,Z)-, biological studies 63-91-2, L-Phenylalanine, biological studies 65-71-4, Thymine 66-22-8, Uracil, biological studies 67-03-8, Thiamine hydrochloride 71-30-7, Cytosine 73-22-3, L-Tryptophan, biological studies 73-24-5, Adenine, biological studies 73-40-5, Guanine 79-81-2, Retinol palmitate 85-61-0, Coenzyme A, biological studies 86-01-1, Guanosine triphosphate 96-26-4, Dihydroxyacetone 98-92-0, Nicotinamide 112-85-6, Behenic acid 113-79-1, Arginine vasopressin 117-39-5, Quercetin 122-32-7, Triolein 123-33-1, Maleic hydrazide 135-16-0, Tetrahydrofolic acid 137-08-6, Pantothenic acid hemicalcium salt 145-42-6, Sodium taurocholate 154-87-0, Cocarboxylase 329-56-6, Arterenol hydrochloride 361-09-1, Sodium cholate 363-24-6, Prostaglandin E2 **463-40-1**, **Linolenic** acid 481-39-0, Juglone 506-21-8, Linolelaidic acid 506-30-9, Arachidic acid 537-40-6, Trilinolein 551-11-1, Prostaglandin F2α 555-43-1, Tristearin 606-68-8 620-64-4, Triarachidin 745-65-3, Prostaglandin E1 863-57-0, Sodium glycocholate 987-65-5, Adenosine triphosphate disodium salt 1105-02-8, Corticosterone-21-sulfate 1184-16-3

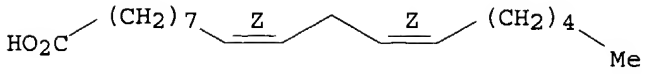
1340-08-5, Vitamin P 1407-47-2, Angiotensin 1731-94-8, Nonadecanoic acid methyl ester 2566-90-7 2644-64-6, Dipalmitoylphosphatidylcholine 2752-99-0, Trierucin 4537-76-2, Distearoylphosphatidylethanolamine 4537-77-3, Dipalmitoylphosphatidylglycerol 4537-78-4, Distearoylphosphatidylglycerol 4539-70-2, Distearoylphosphatidylcholine 4999-79-5, Estradiol-3-sulfate sodium salt 5681-36-7, Dipalmitoylphosphatidylethanolamine 6064-90-0, Heneicosanoic acid methyl ester 6610-25-9, **Arachidonic acid** sodium salt 7235-40-7, β-Carotene 7665-99-8, Cyclic GMP 9001-62-1, Lipase 9002-60-2, Adrenocorticotropic hormone, biological studies 9002-60-2D, Adrenocorticotropic hormone, 1-24 fragment 9002-64-6, Parathyroid hormone 9002-64-6D, Parathyroid hormone, 1-36 fragment 9002-67-9, Luteinizing hormone 9002-68-0, Follicle-stimulating hormone 9002-71-5, Thyrotropic hormone 9002-72-6, Somatotropin 9004-10-8, Insulin, biological studies 9004-61-9, Hyaluronic acid 9005-49-6, Heparin sulfate, biological studies 9007-12-9, Thyrocalcitonin 9007-92-5, Glucagon, biological studies 9015-73-0 9026-43-1, Protein kinase 9041-08-1, Heparin sodium salt **10417-94-4** 10529-43-8, Cholecalciferol sulfate 11000-17-2, Vasopressin 11061-68-0, Human insulin 11128-99-7, Angiotensin II 12629-01-5, Human growth hormone 13487-42-8 14465-68-0 15866-84-9, Adenosine triphosphate calcium salt 18641-57-1, Tribehenin 18656-38-7, Dimyristoylphosphatidylcholine 20255-95-2, Dimyristoylphosphatidylethanolamine 20290-75-9 22251-85-0, Flavin mononucleotide sodium salt 24967-93-9, Chondroitin sulfate A 24967-94-0, Dermatan sulfate 25322-46-7, Chondroitin sulfate C 26536-13-0, Trinonadecanoin 27964-99-4, Poly-D-lysine hydrobromide 28845-86-5, 13,16,19-Docosatrienoic acid, (Z,Z,Z)- 28874-58-0 35121-78-9, Prostaglandin I2 37221-79-7, Vasoactive intestinal peptide 37377-93-8, β-Lipotropin 37377-93-8D, β-Lipotropin, fragment 37839-81-9, Cyclic AMP sodium salt 40245-60-1, Cyclic GMP sodium salt 41598-07-6, Prostaglandin D2 52910-82-4, Aldosterone-21-hemisuccinate 55672-92-9, Coenzyme A sodium salt 59392-49-3, Gastric inhibitory peptide 60617-12-1, β-Endorphin 60617-12-1D, β-Endorphin, fragment 61361-72-6, Dimyristoylphosphatidylglycerol 61849-14-7, Prostaglandin I2 sodium salt 78392-27-5, Cholecalciferol sulfate sodium salt 80380-39-8, Tri-11-eicosenoin 85166-31-0, D-myo-Inositol-1,4,5-triphosphate 92216-45-0, D-myo-Inositol-2,4,5-triphosphate 96012-99-6, Guanosine triphosphate lithium salt 99660-95-4 100775-23-3, Corticosterone-21-sulfate potassium salt 108340-81-4, D-myo-Inositol, 1,4,5-tris(dihydrogen phosphate), hexasodium salt 135271-36-2, D-myo-Inositol-1,4,5-triphosphate potassium salt
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT 7440-70-2, Calcium, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(intracellular, mobilization; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **60-33-3**, 9,12-Octadecadienoic acid (Z,Z)-, biological studies
463-40-1, **Linolenic** acid **10417-94-4**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

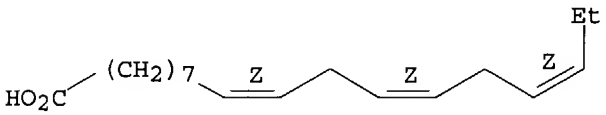
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



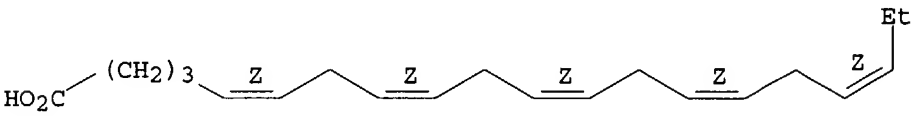
RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 6 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1994:186801 HCAPLUS
DN 120:186801
ED Entered STN: 16 Apr 1994
TI **Lipid** microemulsions for culture media
IN Inlow, Duane
PA USA
SO Can., 35 pp.
CODEN: CAXXA4
DT Patent
LA English
IC ICM C12N005-00
CC 9-11 (Biochemical Methods)
FAN.CNT 1

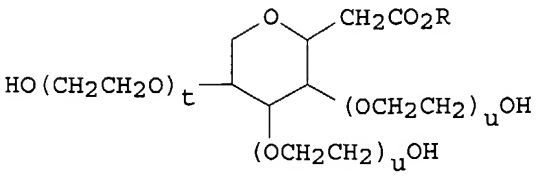
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CA 1315725	A1	19930406	CA 1988-572547	19880720 <--
	EP 377582	A1	19900718	EP 1988-906679	19880720 <--
	EP 377582	B1	19971015		
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	JP 02504225	T2	19901206	JP 1988-506722	19880720 <--
	JP 2788270	B2	19980820		
	AT 159287	E	19971115	AT 1988-906679	19880720 <--
	IL 87195	A1	19940412	IL 1988-87195	19880722 <--
	AU 8943124	A1	19900418	AU 1989-43124	19890913 <--
	US 5372943	A	19941213	US 1993-90568	19930712 <--
PRAI	US 1987-77189	A	19870724 <--		
	WO 1988-US2440		19880720 <--		
	US 1988-248830	A	19880923 <--		
	WO 1989-US3985	A	19890913 <--		
	US 1992-829610	B1	19920130 <--		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES

CA 1315725	ICM	C12N005-00
US 5372943	ECLA	C07K014/53; C12N005/00M; C12N005/00M2; C12N015/866 <--

GI



I

AB **Lipids** are supplied in cell culture media in the form of a microemulsion comprising (1) an aqueous component containing ≥1 emulsifier in water, said emulsifier being selected from **phospholipids** and nontoxic, nonionic, polymeric detergents, and (2) a **lipid** component containing ≥1 **lipid** and ≥1 emulsifier in an organic solvent. The detergent emulsifiers are either (1) block copolymers of propylene oxide and ethylene oxide or (2) polysorbates I (R = (un)saturated C16-20 **fatty acid**; t = 10-30; u = 10-20). Thus, a solution of cod liver oil 100, Tween 80 250, cholesterol 45, and α-tocopherol acetate 20 mg in 10 mL EtOH was filter sterilized and 1 mL was mixed **aseptically** with 10 mL Pluronic F68 for addition to 1 L culture medium for insect cells. Spodoptera frugiperda cells grown in this medium showed a growth rate, maximum cell d., and cell viability comparable to those of cells grown in medium containing 10% serum.

ST **lipid** microemulsion cell culture medium

IT **Phospholipids**, biological studies
RL: BIOL (Biological study)
(as emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)

IT Lecithins
Polymers, biological studies
RL: BIOL (Biological study)
(as nonionic emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)

IT Emulsifying agents
(for **lipid** microemulsions, in serum-free cell culture media)

IT Antioxidants
Reducing agents
Cod-liver oil
RL: BIOL (Biological study)
(**lipid** microemulsions containing, in serum-free cell culture media)

IT Animal tissue culture
(**lipid** microemulsions in serum-free culture media for)

IT **Lipids**, biological studies
RL: BIOL (Biological study)
(microemulsions, in serum-free cell culture media)

IT **Fatty acids**, esters
RL: BIOL (Biological study)
(Me esters, microemulsions, in serum-free cell culture media)

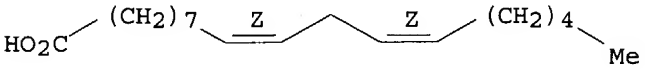
IT Animal cell line
(SF9, **lipid** microemulsions in serum-free culture media for)

IT Vitamins
RL: BIOL (Biological study)
(fat-soluble, microemulsions, in serum-free cell culture media)

IT **Fats and Glyceridic oils**
RL: BIOL (Biological study)
(**fish**-liver, microemulsions, in serum-free cell culture

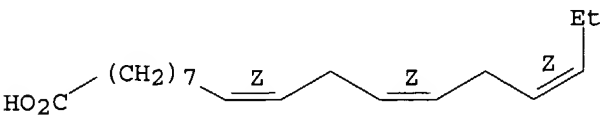
media)
IT Steroids, biological studies
RL: BIOL (Biological study)
(hydroxy, microemulsions, in serum-free cell culture media)
IT Emulsions
(micro-, **lipid**, in serum-free cell culture media)
IT Detergents
(nonionic, polymers, as emulsifying agents in **lipid** microemulsions for serum-free cell culture media)
IT **Fatty acids**, biological studies
RL: BIOL (Biological study)
(**unsatd.**, microemulsions, in serum-free cell culture media)
IT 9005-65-6, Polyoxyethylene sorbitan monooleate 106392-12-5, Ethylene oxide-propylene oxide block copolymer
RL: BIOL (Biological study)
(as nonionic emulsifying agent, in **lipid** microemulsions for serum-free cell culture media)
IT 9005-63-4D, Sorbitan poly(oxy-1,2-ethanediyl) derivs., fatty esters
RL: BIOL (Biological study)
(as nonionic emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)
IT 38098-46-3, Monothioglycerol
RL: BIOL (Biological study)
(**lipid** microemulsions containing, as antioxidant in serum-free cell culture media)
IT 58-95-7, α-Tocopherol acetate
RL: BIOL (Biological study)
(**lipid** microemulsions containing, in serum-free cell culture media)
IT 57-88-5, Cholesterol, biological studies **60-33-3**, **Linoleic acid**, biological studies 112-80-1, Oleic acid, biological studies **463-40-1**, **Linolenic** acid 1406-16-2, Vitamin D 1406-18-4, Vitamin E 11103-57-4, Vitamin A
RL: BIOL (Biological study)
(microemulsions, in serum-free cell culture media)
IT **60-33-3**, **Linoleic acid**, biological studies **463-40-1**, **Linolenic** acid
RL: BIOL (Biological study)
(microemulsions, in serum-free cell culture media)
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 7 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:132151 HCAPLUS

DN 118:132151
ED Entered STN: 30 Mar 1993
TI Therapeutic nutrients containing **glycerides** of branched chain **fatty acids**
IN Sommermeyer, Klaus
PA Fresenius A.-G., Germany
SO Ger. Offen., 6 pp.
CODEN: GWXXBX
DT Patent
LA German
IC ICM A23L001-29
ICS A23K001-00; A23K001-18; A61K031-22; A61K031-23; C07C069-30; C07C067-08
ICA A23L001-035; A61K009-107; A61K009-48; A23L001-0562; A23P001-04
CC 63-6 (**Pharmaceuticals**)
Section cross-reference(s): **17**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 4116004	A1	19921119	DE 1991-4116004	19910516 <--
	DE 4116004	C2	19930930		
	WO 9220241	A1	19921126	WO 1992-EP1049	19920513 <--
	W: JP, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
	EP 584159	A1	19940302	EP 1992-910171	19920513 <--
	EP 584159	B1	19951227		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 06511384	T2	19941222	JP 1992-509184	19920513 <--
	AT 132012	E	19960115	AT 1992-910171	19920513 <--
	ES 2081108	T3	19960216	ES 1992-910171	19920513 <--
	US 5492713	A	19960220	US 1994-150039	19940805 <--
PRAI	DE 1991-4116004		19910516 <--		
	WO 1992-EP1049		19920513 <--		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
DE 4116004	ICM	A23L001-29
	ICS	A23K001-00; A23K001-18; A61K031-22; A61K031-23; C07C069-30; C07C067-08
	ICA	A23L001-035; A61K009-107; A61K009-48; A23L001-0562; A23P001-04

OS MARPAT 118:132151
AB **Glycerides** containing branched **fatty acids**, MeRCH(CH2)m(CH2)nCO2H (R = Me, Et; n = 0 or even number from 2-18; m = 0,1) are used in therapeutic diets for treatment of trauma and **sepsis**, especially uremia. These may be used in oral or parenteral formulations. These **fatty acids** act as a source of branched-chain keto-acids for use in branched chain amino acid biosynthesis to maintain protein synthesis in the liver. The preparation of glyceryl triisovalerinate, glyceryl tri-(3-methylvalerinate), and glyceryl tri-(4-methylvalerinate) by acid-catalyzed esterification is demonstrated. Formulations and dosage forms (capsules) using these **glycerides** are described.
ST **glyceride** branched chain therapeutic nutrient uremia
IT Cottonseed oil
Fats and **Glyceridic** oils
Olive oil
Safflower oil
Soybean oil
Sunflower oil
RL: BIOL (Biological study)
(emulsions containing, in therapeutic nutrients for treatment of uremia)
IT **Glycerides**, biological studies
RL: BIOL (Biological study)

(C6-24, emulsions containing, in therapeutic nutrients for treatment of uremia)
IT Fats and **Glyceridic** oils
RL: BIOL (Biological study)
(animal, emulsions containing, in therapeutic nutrients for treatment of uremia)
IT **Glycerides**, biological studies
RL: BIOL (Biological study)
(branched, therapeutic nutrients containing, for treatment of uremia)
IT **Pharmaceutical** dosage forms
(capsules, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
IT **Pharmaceutical** dosage forms
(emulsions, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
IT Kidney, disease
(failure, treatment of, **glycerides** of branched-chain **fatty acids** in nutritional formulations for)
IT **Fats and Glyceridic oils**
RL: BIOL (Biological study)
(**fish**, emulsions containing, in therapeutic nutrients for treatment of uremia)
IT **Glycerides, biological studies**
RL: BIOL (Biological study)
(**medium-chain**, emulsions containing, in therapeutic nutrients for treatment of uremia)
IT **Glycerides**, biological studies
RL: BIOL (Biological study)
(mono-, emulsions containing, in therapeutic nutrients for treatment of uremia)
IT **Nutrients**
(parenteral, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
IT Animal **nutrition**
(parenteral, for treatment of uremia, **glycerides** of branched-chain **fatty acids** in)
IT 620-63-3P 146369-67-7P 146369-68-8P
RL: PREP (Preparation)
(preparation of, for therapeutic nutrients for treatment of uremia)
IT 105-43-1, 3-Methylvaleric acid 503-74-2, Isovaleric acid 646-07-1, 4-Methylvaleric acid
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, in preparation of **glycerides** for therapeutic nutrients for treatment of uremia)

L78 ANSWER 8 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:100997 HCAPLUS
DN 118:100997
ED Entered STN: 19 Mar 1993
TI Improved metabolic responses to burn injury and endotoxemia with the use of a novel **lipid** source
AU Teo, Tiew Chong
CS Univ. Aberdeen, Aberdeen, UK
SO (**1990**) 210 pp. Avail.: Univ. Microfilms Int., Order No. BRDX94895
From: Diss. Abstr. Int. B 1992, 52(10), 5214
DT Dissertation
LA English
CC 18-5 (Animal **Nutrition**)
AB Unavailable
ST **lipid** enteral diet burn endotoxemia; fish oil **medium chain glyceride** burn
IT Burn

(enteral nutrition in, **lipid** source for)
IT **Lipids**, biological studies
RL: BIOL (Biological study)
(for enteral nutrition in burn injury)
IT **Sepsis and Septicemia**
(**endotoxemia**, from burn injury, **lipid** source for enteral nutrition in)
IT Animal **nutrition**
(enteral, in burn injury, **lipid** source for)
IT **Fats and Glyceridic oils**
RL: BIOL (Biological study)
(**fish**, in enteral nutrition, in burn injury)
IT **Glycerides, biological studies**
RL: BIOL (Biological study)
(**medium-chain**, in enteral nutrition, in burn injury)

L78 ANSWER 9 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1990:421581 HCAPLUS
DN 113:21581
ED Entered STN: 21 Jul 1990
TI Sequential changes in **lipid** metabolism and the **fatty acid** profile in liver **lipids** during fasting and **sepsis**
AU Larsson-Backstroem, Carin; Arrhenius, Eva; Sagge, Kristina; Lindmark, Lars; Paprocki, Jolanta; Svensson, Lennart
CS Dep. Exp. Biol., Kabi Nutr., Stockholm, S-112 87, Swed.
SO Circulatory Shock (**1990**), 30(4), 331-47
CODEN: CRSHAG; ISSN: 0092-6213
DT Journal
LA English
CC 14-3 (Mammalian Pathological Biochemistry)
AB The sequential changes in **lipid** metabolism and in the **fatty acid** profile of liver **lipids** during fasting and **sepsis** were studied. Liver and blood specimens were taken from normally fed rats and from **nonseptically** and **septically** fasted rats at 5, 24, and 48 h. **Sepsis** was induced by injecting live Escherichia coli bacteria i.p. **Sepsis** attenuated the fasting-induced increase in β-hydroxybutyrate and reduced liver and serum **triglycerides** at 5 h. There was a percentage decline in the most abundant **fatty acids** in neutral **lipids**, namely oleic (18:1w9) and **linoleic** (18:2w6) acids. This was seen throughout fasting and **septic** fasting. These results indicate that 18:1w9 and 18:2w6 are used as energy substrates and are oxidized to β-hydroxybutyrate during fasting and mainly to carbon dioxide during **septic** fasting. On the contrary, the most abundant **fatty acids** in **phospholipids**, stearic (18:0), arachidonic (20:4w6), and **docosahexaenoic** (22:6w3) acids, accumulated in neutral **lipids** and in **phospholipids** throughout fasting. However, during **sepsis** this accumulation was reduced in neutral **lipids** and reversed to a level below that in the fed and fasted state in **phospholipids**. These results indicate that a disturbance in membrane integrity and function induced by **septic** fasting may have pathophysiol. consequences for **lipid** metabolism and liver function during **sepsis**.
ST **fatty acid** liver **lipid** metab **sepsis**
IT **Lipids**, biological studies
Phospholipids, biological studies
RL: BIOL (Biological study)
(**fatty acid** profile and metabolism of, sequential changes in, in liver, in fasting and **sepsis**)
IT **Sepsis and Septicemia**

(**lipid** metabolism by liver in fasting and, **fatty acid** profile of liver **lipids** sequential changes in relation to)

IT Starvation
(**lipid** metabolism by liver in **sepsis** and, **fatty acid** profile of liver **lipids** sequential changes in relation to)

IT Liver, metabolism
(**lipid** metabolism by, in fasting and **sepsis**, **fatty acid** profile of liver **lipids** sequential changes in)

IT **Glycerides**, biological studies
RL: BIOL (Biological study)
(of **lipids** and **phospholipids** of liver, in fasting and **sepsis**)

IT **Fatty acids**, biological studies
RL: BIOL (Biological study)
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)

IT 300-85-6, β-Hydroxybutyric acid
RL: BIOL (Biological study)
(of blood serum, in fasting and **sepsis**, **lipid** metabolism by liver in relation to)

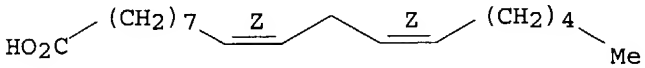
IT 57-88-5, Cholest-5-en-3-ol (3β)-, biological studies
RL: BIOL (Biological study)
(of **lipids** and **phospholipids** of liver, in fasting and **sepsis**)

IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies **60-33-3**, **Linoleic acid**, biological studies 112-80-1, 9-Octadecenoic acid (Z)-, biological studies **463-40-1 506-32-1**, **Arachidonic acid 6217-54-5 10417-94-4**
RL: BIOL (Biological study)
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)

IT **60-33-3**, **Linoleic acid**, biological studies
463-40-1 506-32-1, **Arachidonic acid**
6217-54-5 10417-94-4
RL: BIOL (Biological study)
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)

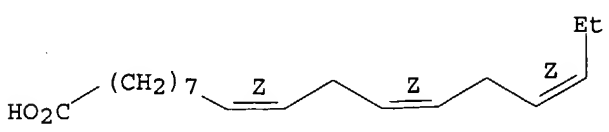
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

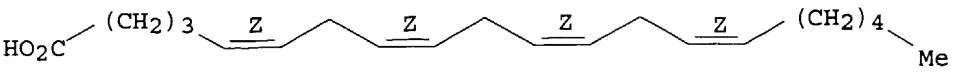
Double bond geometry as shown.



RN 506-32-1 HCAPLUS

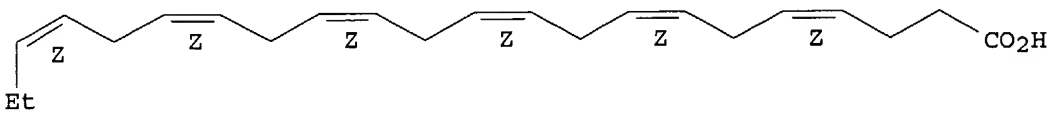
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



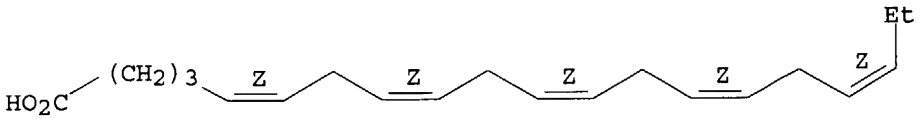
RN 6217-54-5 HCAPLUS
CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 10 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1988:629343 HCAPLUS
DN 109:229343
ED Entered STN: 24 Dec 1988
TI **Lipid** metabolism during starvation and **sepsis** in relation to **fatty acid** profile in liver and α-**linolenic**- and γ -**linolenic acid** -enriched diets
AU Larsson-Backstroem, C.; Paprocki, J.; Lindmark, L.; Svensson, L.
CS Dep. Pharmacol., KabiVitrum AB, Stockholm, S-112 87, Swed.
SO NATO ASI Series, Series A: Life Sciences (**1987**), 139(Lipid Mediators Immunol. Shock), 125-8
CODEN: NALSDJ; ISSN: 0258-1213
DT Journal
LA English
CC 18-5 (Animal **Nutrition**)
Section cross-reference(s): 14
AB In expts. on rats (90 g), fasting for 24 h increased blood free **fatty acids** and β-hydroxybutyrate levels as well as the level of prekallikrein and the platelet count and decreased blood glucose. During fasting, liver **lipid** and **phospholipid** C18:2.**omega.6** and C22:6.**omega.3** were decreased, whereas C20:4.**omega.6** and C22:6.**omega.3** were accumulated in neutral **lipids.** These changes were lower during exptl. **sepsis.** The level of C20:4.**omega.6** in **phospholipids** did not change during fasting, whereas it decreased during **sepsis.** C18:2.**omega.6** and C20:5.**omega.3** in **phospholipids** were reduced during fasting but unaltered in

sepsis. Dietary α- **linolenic** (ALA) and γ-
linolenic (GLA) acids increased the incorporation of .
omega.-3 and ω **-6 fatty**
acids into liver neutral **lipids** and
phospholipids, resp., with a resulting increase in the
unsatn. index. The degree of **unsatn.** following dietary
ALA and GLA was lower during fasting and higher during **sepsis**,
as compared to fed controls. Dietary ALA and GLA reduced liver
triglycerides and enhanced blood free **fatty**
acids, both in fasted and in **septic**-fasted rats. The
level of β-hydroxybutyrate was lower during fasting, but higher
during **sepsis**, compared to the control group. Evidently, there
is a relation between ketogenesis and the degree of **unsatn.** of
liver **lipids.**

ST starvation **lipid** metab blood liver; **sepsis**
lipid metab blood liver; linolenate diet **sepsis**
lipid metab; **fatty acid** liver blood starvation
sepsis

IT Liver, composition
(**fatty acids** of **lipids** and
phospholipids of, starvation and **sepsis** effect on,
dietary α- and γ **-linolenic**
acids in relation to)

IT **Phospholipids**, biological studies
RL: BIOL (Biological study)
(**fatty acids** of, of liver, starvation and
sepsis effect on, dietary α- and γ -
linolenic acids in relation to)

IT **Sepsis and Septicemia**
Starvation
(**lipid** metabolism in, dietary α- and γ -
linolenic acids in relation to)

IT **Lipids**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, starvation and **sepsis** effect on, dietary α-
and γ **-linolenic acids** effect on)

IT **Fatty acids**, biological studies
RL: BIOL (Biological study)
(of blood and liver, starvation and **sepsis** effect on, dietary
α- and γ **-linolenic acids** in
relation to)

IT **Glycerides**, biological studies
RL: BIOL (Biological study)
(of liver, starvation and **sepsis** effect on, dietary α-
and γ **-linolenic acids** in relation
to)

IT Blood sugar
(starvation and **sepsis** effect on, dietary α- and
γ **-linolenic acids** in relation to)

IT **Lipids**, biological studies
RL: BIOL (Biological study)
(neutral, **fatty acids** of, of liver, starvation and
sepsis effect on, dietary α- and γ -
linolenic acids in relation to)

IT **463-40-1**, α **-Linolenic acid**
506-26-3, γ **-Linolenic acid**
RL: BIOL (Biological study)
(**lipid** metabolism in starvation and **sepsis** in relation
to dietary)

IT 300-85-6 9055-02-1, Prekallikrein
RL: BIOL (Biological study)
(of blood, starvation and **sepsis** effect on, dietary α-

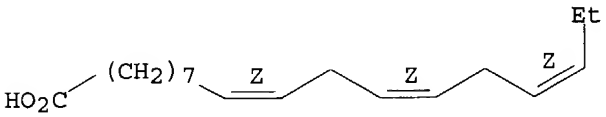
and γ -**linolenic acids** in relation to)

IT **60-33-3**, biological studies **506-32-1 6217-54-5 10417-94-4**
RL: BIOL (Biological study)
(of neutral **lipids** and **phospholipids**, of liver, starvation and **sepsis** effect on, dietary α- and γ -**linolenic acids** in relation to)

IT **463-40-1**, α -**Linolenic acid**
506-26-3, γ -**Linolenic acid**
RL: BIOL (Biological study)
(**lipid** metabolism in starvation and **sepsis** in relation to dietary)

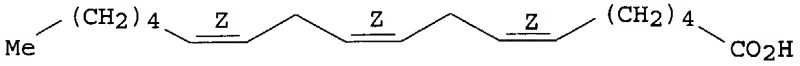
RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 506-26-3 HCAPLUS
CN 6,9,12-Octadecatrienoic acid, (6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

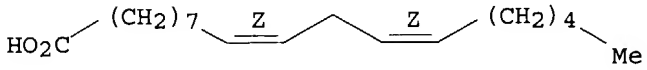
Double bond geometry as shown.



IT **60-33-3**, biological studies **506-32-1 6217-54-5 10417-94-4**
RL: BIOL (Biological study)
(of neutral **lipids** and **phospholipids**, of liver, starvation and **sepsis** effect on, dietary α- and γ -**linolenic acids** in relation to)

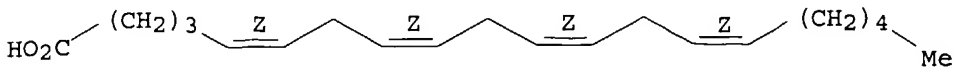
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



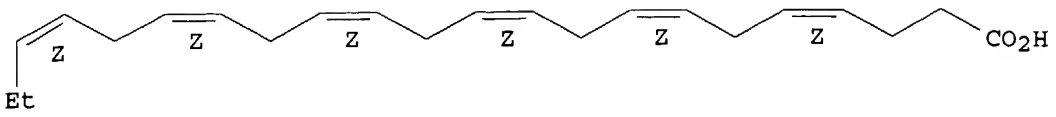
RN 506-32-1 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



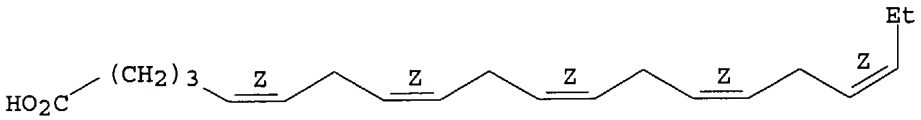
RN 6217-54-5 HCAPLUS
CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

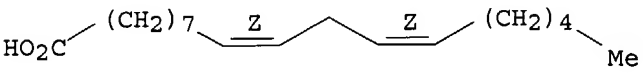
Double bond geometry as shown.



L78 ANSWER 11 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN
AN 1962:452201 HCAPLUS
DN 57:52201
OREF 57:10424h-i,10425a
ED Entered STN: 22 Apr 2001
TI Effects of anaphylaxis on the **lipid** metabolism of guinea pig lung
AU Goadby, P.; Smith, W. G.
CS Sunderland Tech. Coll., UK
SO Biochemical Journal (**1962**), 84, 21P
CODEN: BIJOAK; ISSN: 0264-6021
DT Journal
LA Unavailable
CC 70 (Immunochemistry)
AB Following anaphylaxis induced by exposing sensitized guinea pigs to aerosolized antigen, the following changes in **lipid** content of the lungs were observed: 30% loss of total cholesterol, 15% loss of total **phospholipid** during the 1st 15 min. after shock, and replacement of the lost **lipid** during the subsequent 15 min. Both neutral **lipid** and **phospholipid** fraction had normal **fatty acid** composition 15 min. after shock, but at 30 and 60 min. abnormalities were noted: less stearate and more linoleate in neutral **lipids**, less linoleate and linolenate and more palmitate in **phospholipids.** It is concluded that a loss of cholesterol and **phospholipid** (high-d. lipoprotein), stimulation of cholesterol, **glyceride**, and glycerophosphatide synthesis to replace lost constituents, and replacement of a high proportion of the lung **lipids** with newly synthesized material of abnormal **fatty acid** composition constitute the in vivo changes in **lipid** metabolism. It is suggested the distorted **lipid** metabolism may exert a profound influence on the course of disease processes having an immunological basis.
IT **Phospholipids**
(in lungs, in anaphylaxis)
IT Antibodies
(in ragweed allergy)
IT Lungs
(**lipid** metabolism by, in anaphylaxis)
IT **Anaphylaxis**
(**lipids** in lungs in, metabolism of)
IT **Lipids**

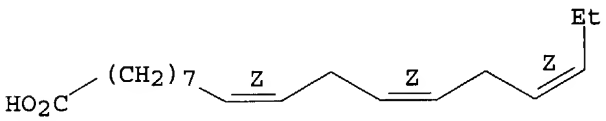
(metabolism of, by lungs in anaphylaxis)
IT 57-10-3, Palmitic acid 57-11-4, Stearic acid **60-33-3**, **Linoleic acid 463-40-1**, **Linolenic** acid
(in lung in anaphylaxis)
IT 57-88-5, Cholesterol
(in lungs, in anaphylaxis)
IT **60-33-3**, **Linoleic acid 463-40-1**, **Linolenic** acid
(in lung in anaphylaxis)
RN 60-33-3 HCAPLUS
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 463-40-1 HCAPLUS
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



=> => fil medline
FILE 'MEDLINE' ENTERED AT 10:52:08 ON 31 AUG 2004

FILE LAST UPDATED: 28 AUG 2004 (20040828/UP). FILE COVERS 1951 TO DATE.

On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD for details. OLDMEDLINE now back to 1951.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See http://www.nlm.nih.gov/mesh/ and http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a description of changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all tot

L109 ANSWER 1 OF 30 MEDLINE on STN
AN 2003420638 MEDLINE
DN PubMed ID: 12897994
TI **Omega-3** vs. **omega-6 lipid** emulsions exert differential influence on neutrophils in **septic shock** patients: impact on plasma **fatty acids** and **lipid** mediator generation.
AU Mayer Konstantin; Fegbeutel Christine; Hattar Katja; Sibelius Ulf; Kramer Hans-Joachim; Heuer Kai-Uwe; Temmesfeld-Wollbruck Bettina; Gokorsch Stephanie; Grimminger Friedrich; Seeger Werner
CS Department of Internal Medicine, Justus Liebig University, Klinikstrasse 36, Giessen, Germany.. Konstantin.Mayer@innere.med.uni-giessen.de

SO Intensive care medicine, (2003 Sep) 29 (9) 1472-81.
Journal code: 7704851. ISSN: 0342-4642.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 200403
ED Entered STN: 20030909
Last Updated on STN: 20040302
Entered Medline: 20040301

AB OBJECTIVE: To compare the effects of a conventional **omega-6 lipid** infusion and a **fish oil** based (**omega-3**) **lipid** infusion for parenteral nutrition on neutrophil function, **lipid** mediators, and plasma free **fatty acids.** DESIGN AND SETTING: Open-label, randomized, pilot study in a university hospital medical intensive care unit and experimental laboratory. PATIENTS AND PARTICIPANTS: Ten patients with **septic shock** and eight healthy controls. INTERVENTIONS: Patients (five per group) requiring parenteral nutrition received intravenously either a **omega-3** or a **omega-6 lipid** emulsion for a 10-day period. MEASUREMENTS AND RESULTS: At baseline levels of plasma free **fatty acids** were elevated several-fold, including high concentrations of the **omega-6 lipid** precursor **arachidonic acid** (AA). Neutrophils isolated from **septic** patients displayed markedly reduced responsiveness to ex vivo stimulation, including **lipid** mediator generation [leukotrienes (LT), PAF], respiratory burst, and phosphoinositide hydrolysis signaling. Under the **omega-6 lipid** infusion regimen abnormalities in plasma free **fatty acids** and impairment of neutrophil functions persisted or worsened. In contrast, a rapid switch in the plasma free **fatty acid** fraction to predominance of the **omega-3** acids **eicosapentaenoic acid** and **docosahexaenoic acid** over AA occurred in response to **omega-3 lipid** infusion. LTB(5), in addition to LTB(4), appeared upon neutrophil stimulation originating from these patients, and neutrophil function was significantly improved in the **omega-3 lipid** group. CONCLUSIONS: **omega-3** vs. **omega-6 lipid** emulsions differentially influence the plasma free **fatty acid** profile with impact on neutrophil functions. **Lipid**-based parenteral nutrition in **septic** patients may thus exert profound influence on sequelae and status of immunocompetence and inflammation.

CT Check Tags: Comparative Study; Female; Human; Male; Support, Non-U.S. Gov't
Adult
Aged
*Fat Emulsions, Intravenous: AD, administration & dosage
***Fatty Acids, Nonesterified: BL, blood**
***Fatty Acids, Omega-3: AD, administration & dosage**
***Fatty Acids, Omega-6: AD, administration & dosage**
Fish Oils: AD, administration & dosage
Leukotrienes: ME, metabolism
Middle Aged
*Neutrophils: ME, metabolism
Phosphatidylinositols: ME, metabolism
Pilot Projects
Platelet Activating Factor: ME, metabolism
Shock, Septic: ME, metabolism

***Shock, Septic: TH, therapy**
Superoxides: ME, metabolism
Thromboxanes: ME, metabolism
RN 11062-77-4 (Superoxides)
CN 0 (Fat Emulsions, Intravenous); 0 (**Fatty Acids**, Nonesterified); 0 (**Fatty Acids, Omega-3**); 0 (**Fatty Acids, Omega-6**); 0 (**Fish Oils**); 0 (Leukotrienes); 0 (Phosphatidylinositols); 0 (Platelet Activating Factor); 0 (Thromboxanes)

L109 ANSWER 2 OF 30 MEDLINE on STN
AN 2003216984 MEDLINE
DN PubMed ID: 12615625
TI Parenteral nutrition with **fish oil** modulates cytokine response in patients with **sepsis.**
AU Mayer Konstantin; Gokorsch Stephanie; Fegbeutel Christine; Hattar Katja; Rosseau Simone; Walmrath Dieter; Seeger Werner; Grimminger Friedrich
CS Medizinische Klinik II, Justus-Liebig-University, Giessen, Germany.. Konstantin.Mayer@innere.med.uni-giessen.de
SO American journal of respiratory and critical care medicine, (2003 May 15) 167 (10) 1321-8.
Journal code: 9421642. ISSN: 1073-449X.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200306
ED Entered STN: 20030513
Last Updated on STN: 20030612
Entered Medline: 20030611
AB Infusion of **fish oil**-based (**n-3**) **lipids** may influence leukocyte function and plasma **lipids** in critical care patients. Twenty-one patients with **sepsis** requiring parenteral nutrition were randomized to receive an **n-3 lipid** emulsion rich in **eicosapentaenoic acid** and **docosahexaenoic acid** or a conventional (**n-6**) **lipid** emulsion (index **fatty acid: arachidonic acid**) for 5 days. The impact on plasma-free **fatty acids**, mononuclear leukocyte cytokine generation, and membrane **fatty acid** composition was examined. Cytokine synthesis by isolated mononuclear leukocyte was elicited by endotoxin. Before the onset of **lipid** infusion therapy, plasma-free **fatty acid** concentrations were greatly increased in **septic** patients, with **arachidonic acid** by far surpassing **eicosapentaenoic acid** and **docosahexaenoic acid**, a feature maintained during conventional **lipid** infusion. Within 2 days of **fish oil** infusion, free **n-3 fatty acids** increased, and the **n-3/n-6** ratio was reversed, with rapid incorporation of **n-3 fatty acids** into mononuclear leukocyte membranes. Generation of proinflammatory cytokines by mononuclear leukocytes was markedly amplified during **n-6** and was suppressed during **n-3 lipid** application. After termination of **lipid** administration, free **n-3 fatty acid** concentrations and mononuclear leukocyte cytokine synthesis returned to preinfusion values. Use of **lipid** infusions might allow us to combine intravenous alimentation with differential impact on inflammatory events and immunologic functions in patients with **sepsis.**
CT Check Tags: Comparative Study; Female; Human; Male; Support, Non-U.S.

Gov't
Critical Illness
Cytokines: AN, analysis
*Cytokines: BI, biosynthesis
Docosahexaenoic Acids: AD, administration & dosage
Eicosapentaenoic Acid: AD, administration & dosage
Fat Emulsions, Intravenous: AD, administration & dosage
***Fish Oils: AD, administration & dosage**
Follow-Up Studies
Leukocytes, Mononuclear: ME, metabolism
*Parenteral Nutrition: MT, methods
Sensitivity and Specificity
Sepsis: DI, diagnosis
***Sepsis: TH, therapy**
Shock, Septic: DI, diagnosis
***Shock, Septic: TH, therapy**
Treatment Outcome
RN **1553-41-9 (Eicosapentaenoic Acid); 25167-62-8 (Docosahexaenoic Acids)**
CN 0 (Cytokines); 0 (Fat Emulsions, Intravenous); 0 (**Fish Oils**)

L109 ANSWER 3 OF 30 MEDLINE on STN
AN 2002492402 MEDLINE
DN PubMed ID: 12353921
TI A **fish oil** emulsion used for parenteral nutrition attenuates monocyte-endothelial interactions under flow.
AU Nohe Boris; Ruoff Heinrich; Johannes Tanja; Zanke Christof; Unertl Klaus; Dieterich Hans-Juergen
CS Department of Anesthesiology and Critical Care, University Hospital Tuebingen, Germany.
SO Shock (Augusta, Ga.), (2002 Sep) 18 (3) 217-22.
Journal code: 9421564. ISSN: 1073-2322.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200312
ED Entered STN: 20021001
Last Updated on STN: 20021213
Entered Medline: 20031218
AB Monocyte adhesion contributes to perfusion abnormalities, tissue damage, and activation of the coagulation system seen during trauma, **shock**, or overwhelming inflammation. This study was performed to determine whether an intravenous **fish oil** emulsion used for parenteral nutrition attenuates monocyte-endothelial interactions under flow and reduces procoagulant activity, measured as tissue factor (TF) expression on adherent monocytes in vitro. Endothelial cell monolayers were incubated with either an intravenous **fish oil** emulsion or a conventional **omega**-6 **lipid** emulsion at 0.05 to 1 mg/ml for 24 h. Six hours following activation with TNFalpha (25 ng/ml), expression of endothelial cell adhesion molecules was measured by flow cytometry. Adhesion of isolated monocytes to pretreated endothelium was examined in a parallel plate flow chamber at a shear stress of 1.5 dynes/cm2. Following perfusion, the cells were cocultured for an additional 4 h and TF expression on monocytes was determined by flow cytometry. In contrast to **omega**-6 **lipids**, **fish oil** down-regulated E-selectin, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1 in a dose-dependent manner. P-selectin, however, remained unchanged. In addition, firm adhesion was reduced to 54%, whereas rolling interactions remained unchanged. **Fish oil** exhibited no effect on the TF expression on cocultured monocytes. We conclude that intravenous

fish oil emulsions reduce both endothelial cell adhesion molecule expression and monocyte adhesion. However, under postcapillary flow conditions, rolling interactions via P-selectin remain unaltered. The functional importance of this effect is illustrated by the corresponding upregulation of TF in response to residual monocyte-endothelial interactions.

CT Check Tags: Human; Support, Non-U.S. Gov't
Cell Adhesion: DE, drug effects
Cell Adhesion Molecules: ME, metabolism
Cells, Cultured
Coculture
Cytokines: PD, pharmacology
*Emulsions: PD, pharmacology
*Endothelium: CY, cytology
*Endothelium: DE, drug effects
Endothelium: ME, metabolism
***Fish Oils: PD, pharmacology**
*Monocytes: CY, cytology
*Monocytes: DE, drug effects
Monocytes: ME, metabolism
*Parenteral Nutrition
Thromboplastin: ME, metabolism

RN 9035-58-9 (Thromboplastin)

CN 0 (Cell Adhesion Molecules); 0 (Cytokines); 0 (Emulsions); 0 (**Fish Oils**)

L109 ANSWER 4 OF 30 MEDLINE on STN

AN 2002162753 MEDLINE

DN PubMed ID: 11895156

TI Parenteral nutrition with n-3 **lipids** in **sepsis.**

AU Mayer K; Grimm H; Grimminger F; Seeger W

CS Medizinische Klinik II der Justus-Liebig-Universitat Giessen, Germany.. Konstantin.mayer@innere.med.unigiessen.de

SO British journal of nutrition, (2002 Jan) 87 Suppl 1 S69-75. Ref: 67
Journal code: 0372547. ISSN: 0007-1145.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200204

ED Entered STN: 20020317
Last Updated on STN: 20020419
Entered Medline: 20020418

AB Dietary supplements of **n-3 fatty acids** have long been used to influence chronic inflammatory disorders. Recent studies with an immune-enhancing diet partly based on **n-3 fatty acids** report beneficial effects in patients with acute hyper-inflammatory diseases, such as the **sepsis** syndrome or adult respiratory distress syndrome (ARDS). The possible suppression of exaggerated leucocyte activity, the improvement of microcirculatory events, as well as the opportunity to administer intravenous **lipids** enriched in **n-3 fatty acids** signal the possibility of a combination of parenteral caloric support and pharmacological intervention. Using parenteral administration of **fish oil**-based **lipids**, a new rapid and highly effective anti-inflammatory agent may allow the option to alter the immune status in hyper-inflammatory diseases such as **sepsis** and ARDS.

CT Check Tags: Human
***Fatty Acids, Omega-3: TU, therapeutic use**
Inflammation: TH, therapy

*Parenteral Nutrition: MT, methods
Respiratory Distress Syndrome, Adult: TH, therapy
Sepsis Syndrome: PP, physiopathology
***Sepsis Syndrome: TH, therapy**
CN 0 (**Fatty Acids, Omega-3**)

L109 ANSWER 5 OF 30 MEDLINE on STN
AN 2001634858 MEDLINE
DN PubMed ID: 11240338
TI Parenteral supplementation with a **fish-oil** emulsion prolongs survival and improves rat lymphocyte function during **sepsis.**
CM Comment in: Nutrition. 2001 Feb;17(2):158-60. PubMed ID: 11240346
AU Lanza-Jacoby S; Flynn J T; Miller S
CS Department of Surgery, Thomas Jefferson University, Jefferson Medical College, Philadelphia, Pennsylvania 19107, USA.. susan.lanza-jacoby@mail.tju.edu
SO Nutrition (Burbank, Los Angeles County, Calif.), (2001 Feb) 17 (2) 112-6. Journal code: 8802712. ISSN: 0899-9007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200111
ED Entered STN: 20011105
Last Updated on STN: 20011105
Entered Medline: 20011101
AB Nutritional intervention with **omega-3 fatty acids** during trauma and infection has been shown to improve the clinical outcome of patients and the survival rate in laboratory animals. This study evaluated the effects of parenteral administration of **lipid** emulsions containing **fish oil** (FO) or soybean oil (SBO) on survival and T-lymphocyte response during **sepsis.** Male Sprague-Dawley rats (250-275 g) were prepared for parenteral feeding 4 d before inducing **sepsis** by cecal ligation and puncture (CLP). Standard resuscitation was provided with normal saline. Thirty minutes after completing CLP, sham control or CLP rats were infused continuously with saline or a parenteral diet containing SBO or a 1:1 FO:SBO emulsion. The survival rate was significantly improved in rats receiving the FO-supplemented diet, with 50% alive by 120 h in comparison with the saline-infused, chow-fed rats (0% alive by 120 h) or the SBO-fed rats (12% alive at 120 h). The T-lymphocyte response was evaluated at 24 h after CLP. **Sepsis** led to a decline in lymphocyte proliferation in rats infused with saline or the SBO emulsion, which was associated with a greater release of splenocyte interleukin-10, transforming growth factor-beta and prostaglandin E2. Administering the 1:1 FO:SBO parenteral diet during **sepsis** improved the survival rate and prevented the **sepsis**-induced suppression of lymphocyte proliferation and interleukin-2 release. The FO effect on lymphocyte function was associated with decreased splenocyte release of transforming growth factor-beta and prostaglandin E2.
CT Check Tags: Male
Animals
Cecum: SU, surgery
Dietary Supplements
Dinoprostone: BI, biosynthesis
*Fat Emulsions, Intravenous: AD, administration & dosage
Fat Emulsions, Intravenous: CH, chemistry
***Fish Oils: AD, administration & dosage**
Fish Oils: PD, pharmacology
Interleukin-10: SE, secretion
Interleukin-2: SE, secretion
Ligation

Lymphocyte Activation
*Parenteral Nutrition
Punctures
Rats
Rats, Sprague-Dawley
Sepsis: IM, immunology
Sepsis: MO, mortality
***Sepsis: TH, therapy**
Soybean Oil: AD, administration & dosage
Soybean Oil: PD, pharmacology
Survival Analysis
*T-Lymphocytes: IM, immunology
Transforming Growth Factor beta: BL, blood
Transforming Growth Factor beta: SE, secretion
RN 130068-27-8 (Interleukin-10); 363-24-6 (Dinoprostone); 8001-22-7 (Soybean Oil)
CN 0 (Fat Emulsions, Intravenous); 0 (**Fish Oils**); 0 (Interleukin-2); 0 (Transforming Growth Factor beta)

L109 ANSWER 6 OF 30 MEDLINE on STN
AN 2001608625 MEDLINE
DN PubMed ID: 11685569
TI Immunonutrition--supplementary amino acids and **fatty acids** ameliorate immune deficiency in critically ill patients.
AU Grimm H; Kraus A
CS Department of General and Thoracic Surgery, Justus Liebig University, Rudolf-Buchheim-Strasse 7, 35385 Giessen, Germany.. helmut.grimm@chiru.med.uni-giessen.de
SO Langenbeck's archives of surgery / Deutsche Gesellschaft fur Chirurgie, (2001 Aug) 386 (5) 369-76. Ref: 70
Journal code: 9808285. ISSN: 1435-2443.
CY Germany: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20011102
Last Updated on STN: 20020123
Entered Medline: 20011205
AB BACKGROUND: Immunonutrition with **omega-3 fatty acids** and the "conditionally essential" amino acids arginine, glutamine, cysteine, and taurine can enhance the immune response in critically ill patients. This is due to the immunomodulating properties of these nutrients. Immunonutrition is especially important when a patient's immune response is compromised, as is the case post-operatively or after trauma. Immune deficiency is severely aggravated in **sepsis** and the systemic inflammatory response syndrome (SIRS). The resulting metabolic stress is characterized by glycolysis, lipolysis, and proteolysis, which may escalate to an hypercatabolic response or "autocannabilism." Catabolic metabolism results in insufficiency of both specific and unspecific immunocompetent cells. CONCLUSIONS: Immunonutrition should be started early in such patients for an optimal beneficial effect, preferably via the enteral route. It should include **medium chain** and long chain **triglycerides**, polyunsaturated **omega-3** and **omega-6** **fatty acids** (in the ratio 1:2), olive oil, and conventional amino acid preparations supplemented with the conditionally essential amino acids arginine, glutamine, cysteine, and taurine.
CT Check Tags: Human
*Adjuvants, Immunologic: TU, therapeutic use
*Amino Acids, Essential: TU, therapeutic use

Date no good

*Critical Illness
*Dietary Supplements
Endotoxins: BL, blood
Enteral Nutrition: MT, methods
***Fatty Acids, Omega-3: TU, therapeutic use**
*Immunocompromised Host: PH, physiology
*Immunologic Deficiency Syndromes: DH, diet therapy
Immunologic Deficiency Syndromes: ET, etiology
Immunologic Deficiency Syndromes: ME, metabolism
Lipids: ME, metabolism
Proteins: DE, drug effects
Proteins: ME, metabolism
Signal Transduction: DE, drug effects
CN 0 (Adjuvants, Immunologic); 0 (Amino Acids, Essential); 0 (Endotoxins); 0 (**Fatty Acids, Omega-3**); 0 (**Lipids**); 0 (Proteins)

L109 ANSWER 7 OF 30 MEDLINE on STN
AN 2001140272 MEDLINE
DN PubMed ID: 11133172
TI Beneficial effect(s) of **n-3 fatty acids** in cardiovascular diseases: but, why and how?.
CM Erratum in: Prostaglandins Leukot Essent Fatty Acids 2001 Jan;64(1):74
AU Das U N
CS EFA Sciences LLC, 1420 Providence Highway, Norwood, MA 02062, USA.. undurti@hotmail.com
SO Prostaglandins, leukotrienes, and essential fatty acids, (2000 Dec) 63 (6) 351-62. Ref: 143
Journal code: 8802730. ISSN: 0952-3278.
CY Scotland: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20011018
Entered Medline: 20010308
AB Low rates of coronary heart disease was found in Greenland Eskimos and Japanese who are exposed to a diet rich in **fish oil.** Suggested mechanisms for this cardio-protective effect focused on the effects of **n-3 fatty acids** on eicosanoid metabolism, inflammation, beta oxidation, endothelial dysfunction, cytokine growth factors, and gene expression of adhesion molecules; But, none of these mechanisms could adequately explain the beneficial actions of **n-3 fatty acids.** One attractive suggestion is a direct cardiac effect of **n-3 fatty acids** on arrhythmogenesis. **N-3 fatty acids** can modify Na+ channels by directly binding to the channel proteins and thus, prevent ischemia-induced ventricular fibrillation and sudden cardiac death. Though this is an attractive explanation, there could be other actions as well. **N-3 fatty acids** can inhibit the synthesis and release of pro-inflammatory cytokines such as tumor necrosis factoralpha (TNFalpha) and interleukin-1 (IL-1) and IL-2 that are released during the early course of ischemic heart disease. These cytokines decrease myocardial contractility and induce myocardial damage, enhance the production of free radicals, which can also suppress myocardial function. Further, **n-3 fatty acids** can increase parasympathetic tone leading to an increase in heart rate variability and thus, protect the myocardium against ventricular arrhythmias. Increased parasympathetic tone and

acetylcholine, the principle vagal neurotransmitter, significantly attenuate the release of TNF, IL-1beta, IL-6 and IL-18. Exercise enhances parasympathetic tone, and the production of anti-inflammatory cytokine IL-10 which may explain the beneficial action of exercise in the prevention of cardiovascular diseases and diabetes mellitus. TNFalpha has neurotoxic actions, where as **n-3 fatty acids** are potent neuroprotectors and brain is rich in these **fatty acids.** Based on this, it is suggested that the principle mechanism of cardioprotective and neuroprotective action(s) of **n-3 fatty acids** can be due to the suppression of TNFalpha and IL synthesis and release, modulation of hypothalamic-pituitary-adrenal anti-inflammatory responses, and an increase in acetylcholine release, the vagal neurotransmitter. Thus, there appears to be a close interaction between the central nervous system, endocrine organs, cytokines, exercise, and dietary **n-3 fatty acids.** This may explain why these **fatty acids** could be of benefit in the management of conditions such as **septicemia** and **septic shock**, Alzheimer's disease, Parkinson's disease, inflammatory bowel diseases, diabetes mellitus, essential hypertension and atherosclerosis.

CT Check Tags: Human
Acetylcholine: PH, physiology
Animals
Arrhythmia: EP, epidemiology
Arrhythmia: PC, prevention & control
Brain: PP, physiopathology
Cardiovascular Diseases: DH, diet therapy
Cardiovascular Diseases: EP, epidemiology
*Cardiovascular Diseases: PC, prevention & control
Cell Adhesion Molecules: BI, biosynthesis
Cell Adhesion Molecules: GE, genetics
Cell Division: DE, drug effects
Clinical Trials
Cohort Studies
Cytokines: ME, metabolism
Dietary Fats: AD, administration & dosage
*Dietary Fats: PD, pharmacology
Dietary Fats: TU, therapeutic use
Eicosanoids: ME, metabolism
Endothelium, Vascular: DE, drug effects
Endothelium, Vascular: ME, metabolism
Exercise
Fatty Acids, Omega-3: AD, administration & dosage
***Fatty Acids, Omega-3: PD, pharmacology**
Fatty Acids, Omega-3: TU, therapeutic use
Fatty Acids, Unsaturated: ME, metabolism
Fish Oils: AD, administration & dosage
***Fish Oils: PD, pharmacology**
Fish Oils: TU, therapeutic use
Gene Expression Regulation: DE, drug effects
Greenland: EP, epidemiology
Heart: DE, drug effects
Hemostasis: DE, drug effects
Hypothalamo-Hypophyseal System: DE, drug effects
Hypothalamo-Hypophyseal System: PP, physiopathology
Inflammation: DT, drug therapy
Inflammation: ME, metabolism
Inflammation: PC, prevention & control
Inuits
Japan: EP, epidemiology
Lipids: ME, metabolism
Models, Biological
Myocardium: ME, metabolism

Oxidation-Reduction
Oxidative Stress
Parasympathetic Nervous System: DE, drug effects
Pituitary-Adrenal System: DE, drug effects
Pituitary-Adrenal System: PP, physiopathology
Rats
Sodium Channels: DE, drug effects
Vagus Nerve: PP, physiopathology
RN 51-84-3 (Acetylcholine)
CN 0 (Cell Adhesion Molecules); 0 (Cytokines); 0 (Dietary Fats); 0 (Eicosanoids); 0 (**Fatty Acids, Omega-3**); 0 (**Fatty Acids**, Unsaturated); 0 (**Fish Oils**); 0 (**Lipids**); 0 (Sodium Channels)

L109 ANSWER 8 OF 30 MEDLINE on STN
AN 2001028371 MEDLINE
DN PubMed ID: 10850941
TI **Fish oil** modulates macrophage P44/P42 mitogen-activated protein kinase activity induced by lipopolysaccharide.
AU Lo C J; Chiu K C; Fu M; Chu A; Helton S
CS Department of Surgery, University of California, Los Angeles, USA.. clo@mednet.ucla.edu
SO JPEN. Journal of parenteral and enteral nutrition, (2000 May-Jun) 24 (3) 159-63.
Journal code: 7804134. ISSN: 0148-6071.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001121
AB BACKGROUND: Mitogen-activated protein kinase (MAPK) cascades represent a major signal system to transduce extracellular signals into cellular responses. Overactivity of MAPK has been implicated in the development of many diseases, including cancer and **sepsis.** This study investigated the hypothesis that **fish oil** altered the membrane phospholipid composition and modulated MAPK activity. METHODS: RAW 264.7 cells, a mouse macrophage (Mphi) cell line, were grown in **eicosapentaenoic acid** (EPA)-rich media (114 micromol/L) for 48 hours. Mphi were washed and exposed to Escherichia coli lipopolysaccharide (LPS; 1 microg/mL) for 10 minutes. Both total and activated (phosphorylated) portions of MAPK (P44 and P42) were determined by Western blot assays. AP-1 transcription factor activity was determined by electrophoretic mobility gel shift assays (EMSA). Mphi tumor necrosis factor (TNF) mRNA expression was measured by Northern blot assays. RESULTS: LPS stimulation induced RAW cell phosphorylation of P44/P42. In contrast, RAW cells grown in EPA-rich media had less P44/P42 activation in the presence of LPS. Total P44/P42 were not affected by EPA or LPS. Similarly, EPA also inhibited AP-1 activity. Inhibition of P44/P42 activity with PD98059 reduced both AP-1 activity and TNF mRNA expression of LPS-stimulated Mphi. CONCLUSIONS: Our data suggest that **fish oil** regulates macrophage proinflammatory gene activation, at least in part, by modulating the MAPK activity.
CT Check Tags: Support, Non-U.S. Gov't
Animals
Arachidonic Acids
Blotting, Western
Cell Line
Down-Regulation
Electrophoresis
Escherichia coli

***Fish Oils: PD, pharmacology**
Lipopolysaccharides: PD, pharmacology
Macrophages: DE, drug effects
Macrophages: EN, enzymology
*Macrophages: PH, physiology
Membrane Lipids: CH, chemistry
Mice
Mitogen-Activated Protein Kinases: DE, drug effects
*Mitogen-Activated Protein Kinases: ME, metabolism
RNA, Messenger
Tumor Necrosis Factor: BI, biosynthesis
Tumor Necrosis Factor: GE, genetics
CN 0 (**Arachidonic Acids**); 0 (**Fish Oils**); 0 (Lipopolysaccharides); 0 (Membrane **Lipids**); 0 (RNA, Messenger); 0 (Tumor Necrosis Factor); EC 2.7.1.37 (Mitogen-Activated Protein Kinases)

L109 ANSWER 9 OF 30 MEDLINE on STN
AN 2000223123 MEDLINE
DN PubMed ID: 10758365
TI Effects of parenteral infusion with **fish-oil** or safflower-oil emulsion on hepatic **lipids**, plasma amino acids, and inflammatory mediators in **septic** rats.
CM Comment in: Nutrition. 2000 Apr;16(4):308-9. PubMed ID: 10758371
AU Chao C Y; Yeh S L; Lin M T; Chen W J
CS Institute of Nutrition and Health Science, Taipei Medical College, Taipei, Taiwan, People's Republic of China.
SO Nutrition (Burbank, Los Angeles County, Calif.), (2000 Apr) 16 (4) 284-8. Journal code: 8802712. ISSN: 0899-9007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
ED Entered STN: 20000720
Last Updated on STN: 20000720
Entered Medline: 20000710
AB This study was designed to investigate the effects of preinfusion with total parenteral nutrition (TPN) using **fish-oil** (FO) versus safflower-oil (SO) emulsion as fat sources on hepatic **lipids**, plasma amino-acid profiles, and inflammatory-related mediators in **septic** rats. Normal rats, with internal jugular catheters, were assigned to two different groups and received TPN. TPN provided 300 kcal. kg(-1). d(-1), with 40% of the non-protein energy as fat. All TPN solutions were isonitrogenous and identical in nutrient composition except for the fat emulsion, which was made of SO or FO. After receiving TPN for 6 d, each group of rats was further divided into control and **sepsis** subgroups. **Sepsis** was induced by cecal ligation and puncture; control rats received sham operation. All rats were classified into four groups as follows: FO control group (FOC; n = 7), FO **sepsis** group (FOS; n = 8), SO control group (SOC; n = 8), and SO **sepsis** group (SOS; n = 9). The results of the study demonstrated that plasma concentrations of triacylglycerol and non-esterified **fatty acids** did not differ between the FO and SO groups, regardless of whether the animals were **septic.** SOS had significantly higher total **lipids** and cholesterol content in the liver than did the SOC group. The FOS group, however, showed no difference from the FOC group. Plasma leucine and isoleucine levels were significantly lower in the SOS group than in the SOC group, whereas no difference in these two amino acids was observed between the FOC and FOS groups. Plasma arginine levels were significantly lower in both **septic** groups than in the groups without **sepsis** when either FO or SO was infused. Plasma glutamine levels, however, did

not differ across groups. No differences in interleukin-1beta, interleukin-6, tumor necrosis factor-alpha, or leukotriene B(4) concentrations in peritoneal lavage fluid were observed between the two **septic** groups. These results suggest that catabolic reaction in **septic** rats preinfused with FO is not as obvious as those preinfused with SO. Compared with SO emulsion, TPN with FO emulsion prevents liver fat accumulation associated with **sepsis.** However, parenterally administered FO had no beneficial effect in lowering cytokines and LTB(4) levels in peritoneal lavage fluid in **septic** rats induced by cecal ligation and puncture.

CT Check Tags: Male; Support, U.S. Gov't, Non-P.H.S.
*Amino Acids: BL, blood
Animals
*Cytokines: BL, blood
Disease Models, Animal
Fat Emulsions, Intravenous: AD, administration & dosage
***Fish Oils: AD, administration & dosage**
Lipids: BL, blood
***Lipids: ME, metabolism**
*Liver: ME, metabolism
*Parenteral Nutrition, Total
Rats
Rats, Wistar
*Safflower Oil: AD, administration & dosage
Sepsis: BL, blood
Sepsis: TH, therapy

RN 8001-23-8 (Safflower Oil)
CN 0 (Amino Acids); 0 (Cytokines); 0 (Fat Emulsions, Intravenous); 0 (**Fish Oils**); 0 (**Lipids**)

L109 ANSWER 10 OF 30 MEDLINE on STN
AN 2000207435 MEDLINE
DN PubMed ID: 10743032
TI [Immunonutrition with **omega-3-fatty acids.** Are new anti-inflammatory strategies in sight?]. Immunonutrition mit **Omega-3** Fettsauren. Sind neue anti-inflammatorische Strategien in Sicht?.
AU Heller A; Koch T
CS Klinik und Poliklinik fur Anasthesiologie und Intensivtherapie, Universitatsklinikum Carl Gustav Carus, Technischen Universitat Dresden.. heller-a@rcs.urz.tu-dresden.de
SO Zentralblatt fur Chirurgie, (2000) 125 (2) 123-36. Ref: 126
Journal code: 0413645. ISSN: 0044-409X.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 200005
ED Entered STN: 20000512
Last Updated on STN: 20000512
Entered Medline: 20000504
AB In the early phase of **sepsis** and SIRS an overwhelming activation of humoral and cellular mediator systems can alter vascular resistance and causes capillary leakage increasing the risk of organ dysfunction. **omega-6-arachidonic acid** is released from **lipid** pools of cellular membranes during inflammation and is metabolized to pro-inflammatory prostaglandins and leukotriens, which are key mediators in the pathogenesis of organ dysfunction. **omega -3-eicosapentaenoic acid**-derived **lipid** mediators present altered biologic effects. Thus, **omega-3-fatty acid** application

enables anti-inflammatory intervention on the level of **lipid** mediators. The current article reviews experimental and clinical data on **omega-3-fatty acids.** Besides the decrease of pro-inflammatory mediators, **fish oil** supplementation lowered post operative infection rates and showed a tendency to reduce hospital stay in surgical patients. It is believed that the decreased formation of LTB4 and TXA2 during **sepsis** after administration of **omega-3-fatty acids** accounts for improved microcirculatory perfusion and declined lactate acidosis.

CT Check Tags: Human
Animals
Capillary Leak Syndrome: IM, immunology
Capillary Leak Syndrome: TH, therapy
English Abstract
***Fatty Acids, Omega-3: AD, administration & dosage**
Inflammation Mediators: BL, blood
Intensive Care
Sepsis Syndrome: IM, immunology
***Sepsis Syndrome: TH, therapy**
Treatment Outcome

CN 0 (**Fatty Acids, Omega-3**); 0 (Inflammation Mediators)

L109 ANSWER 11 OF 30 MEDLINE on STN
AN 1998222123 MEDLINE
DN PubMed ID: 9561339
TI **Lipid** mediators in inflammatory disorders.
AU Heller A; Koch T; Schmeck J; van Ackern K
CS Department of Anaesthesiology and Intensive Care Medicine, University of Dresden, Germany.. heller@rumms.uni-mannheim.de
SO Drugs, **(1998 Apr)** 55 (4) 487-96. Ref: 46
Journal code: 7600076. ISSN: 0012-6667.
CY New Zealand
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199806
ED Entered STN: 19980625
Last Updated on STN: 19980625
Entered Medline: 19980618
AB During the past few decades, intensive collaborative research in the fields of chronic and acute inflammatory disorders has resulted in a better understanding of the pathophysiology and diagnosis of these diseases. Modern therapeutic approaches are still not satisfactory and **shock, sepsis** and multiple organ failure remain the great challenge in intensive care medicine. However, the treatment of inflammatory diseases like rheumatoid arthritis, ulcerative colitis or psoriasis also represents an unresolved problem. Many factors contribute to the complex course of inflammatory reactions. Microbiological, immunological and toxic agents can initiate the inflammatory response by activating a variety of humoral and cellular mediators. In the early phase of inflammation, excessive amounts of interleukins and **lipid** -mediators are released and play a crucial role in the pathogenesis of organ dysfunction. **Arachidonic acid** (AA), the mother substance of the pro-inflammatory eicosanoids, is released from membrane phospholipids in the course of inflammatory activation and is metabolised to prostaglandins and leukotrienes. Various strategies have been evaluated to control the excessive production of **lipid** mediators on different levels of biochemical pathways, such as inhibition of phospholipase A2, the trigger enzyme for release of AA, blockade of

cyclooxygenase and lipoxygenase pathways and the development of receptor antagonists against platelet activating factor and leukotrienes. Some of these agents exert protective effects in different inflammatory disorders such as **septic** organ failure, rheumatoid arthritis or asthma, whereas others fail to do so. Encouraging results have been obtained by dietary supplementation with long chain **omega-3 fatty acids** like **eicosapentaenoic acid** (EPA). In states of inflammation, EPA is released to compete with AA for enzymatic metabolism inducing the production of less inflammatory and chemotactic derivatives.

CT Check Tags: Human
Animals
***Arachidonic Acid: ME, metabolism**
Cyclooxygenase Inhibitors: TU, therapeutic use
Fatty Acids, Omega-3: TU, therapeutic use
*Inflammation: ME, metabolism
Inflammation: TH, therapy
*Inflammation Mediators: ME, metabolism
Leukotriene Antagonists
***Lipids: ME, metabolism**
Lipoxygenase Inhibitors: TU, therapeutic use
Phospholipases A: AI, antagonists & inhibitors
Phospholipids: ME, metabolism
Platelet Activating Factor: AI, antagonists & inhibitors
Prostaglandins: ME, metabolism

RN **506-32-1 (Arachidonic Acid)**

CN 0 (Cyclooxygenase Inhibitors); 0 (**Fatty Acids, Omega-3**); 0 (Inflammation Mediators); 0 (Leukotriene Antagonists); 0 (**Lipids**); 0 (Lipoxygenase Inhibitors); 0 (Phospholipids); 0 (Platelet Activating Factor); 0 (Prostaglandins); EC 3.1.1.- (Phospholipases A)

L109 ANSWER 12 OF 30 MEDLINE on STN

AN 1998219122 MEDLINE

DN PubMed ID: 9558431

TI [Pharmacologic aspects of polyunsaturated **fatty acids** in parenteral nutrition].
Pharmakologische Aspekte von mehrfach ungesattigten Fettsauren in der parenteralen Ernahrung.

AU Heller A; Koch T

CS Institut fur Anasthesiologie und Operative Intensivmedizin, Fakultat fur Klinische Medizin Mannheim, Universitat Heidelberg.. heller@rumms.uni-mannheim.de

SO Anasthesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie : AINS, **(1998 Feb)** 33 (2) 77-87. Ref: 61
Journal code: 9109478. ISSN: 0939-2661.

CY GERMANY: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA German

FS Priority Journals

EM 199806

ED Entered STN: 19980625
Last Updated on STN: 19980625
Entered Medline: 19980616

AB Despite immense progress in intensive-care medicine, mortality rates of 30-70% in **sepsis** and SIRS are still an unresolved problem. Particularly the failure of respiratory and other vital functions is a major cause of death. Besides infectious stimuli (viruses, bacteria, fungi) a variety of non-infectious triggers (tissue damage, immune complexes, complement activation, etc.) can initiate the development of organ failure. These inflammatory reactions aim physiologically towards

inactivation and removal of the stimulating agents as well as the induction of reparative processes. In states of prolonged activation of humoral and cellular mediator systems the natural host defence mechanisms react in an uncontrolled manner causing tissue damage and organ failure. So far there are no efficient therapeutic strategies to influence these complex inflammatory reactions. In the development of SIRS and **sepsis**, pro-inflammatory **lipid** mediators play a crucial role. **Omega-3-fatty acids** (**omega-3**-PUFAs) have shown anti-inflammatory and antithrombotic properties in a great number of experimental and clinical studies. These effects seem to be related to the uptake of **eicosapentaenoic acid** (EPA) into cellular membrane **lipid** pools and its subsequent metabolisation. After inflammatory activation EPA is released besides **arachidonic acid** (AA) and competes with AA for metabolisation via the cyclo- and lipoxygenase pathway. Compared to AA the derivatives of EPA have less pro-inflammatory and chemotactic characteristics. With regard to prophylactic and therapeutic consequences it appears reasonable to supplement **omega-3**-PUFAs to attenuate the inflammatory response by modulating the generation of **lipid** mediators during inflammation.

CT Check Tags: Human
Animals
Eicosanoids: PD, pharmacology
English Abstract
*Fat Emulsions, Intravenous: PD, pharmacology
***Fatty Acids, Omega-3: PD, pharmacology**
Inflammation Mediators: BL, blood
Multiple Organ Failure: IM, immunology
***Sepsis Syndrome: IM, immunology**

CN 0 (Eicosanoids); 0 (Fat Emulsions, Intravenous); 0 (**Fatty Acids, Omega-3**); 0 (Inflammation Mediators)

L109 ANSWER 13 OF 30 MEDLINE on STN
AN 97239214 MEDLINE
DN PubMed ID: 9084876
TI Reevaluation of the effect of a high alpha-linolenate and a high linoleate diet on antigen-induced antibody and anaphylactic responses in mice.
AU Oh-hashi K; Watanabe S; Kobayashi T; Okuyama H
CS Department of Biological Chemistry, Faculty of Pharmaceutical Sciences, Nagoya City University, Japan.
SO Biological & pharmaceutical bulletin, **(1997 Mar)** 20 (3) 217-23.
Journal code: 9311984. ISSN: 0918-6158.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199706
ED Entered STN: 19970630
Last Updated on STN: 19970630
Entered Medline: 19970619
AB Previously, we reported that a high alpha-linolenate [18:3(n-3)] diet compared with a high linoleate [18:2(n-6)] diet suppressed the anti-egg albumin (EA) immunoglobulin E (IgE) antibody response in mice. Because of relatively high background values obtained with the method used previously, we used an improved ELISA and once again determined serum IgE levels. In contrast to our previous results, the serum level of anti-dinitrophenyl specific (anti-DNP) as well as total IgE in mice immunized with DNP-antigen was slightly but significantly higher in the high alpha-linolenate diet group than in the high linoleate diet group. Anti-DNP IgG1 and IgG2a antibody responses were not significantly different in mice fed these diets. Indomethacin administration during immunization tended to enhance the IgE antibody responses. The mortality

of mice from antigen-induced anaphylactic **shock** was significantly lower in the high alpha-linolenate diet group than in the high linoleate diet group; however, there was no difference between the groups in terms of vascular permeability and histamine levels. Thus, the high alpha-linolenate diet enhances the IgE antibody response slightly without affecting either the IgG antibody response, vascular permeability or histamine release. The high alpha-linolenate diet possibly suppresses anaphylactic **shock** by reducing the synthesis of **lipid** mediators such as eicosanoids and platelet-activating factor.

CT Check Tags: Female
Anaphylaxis: MO, mortality
*Anaphylaxis: PP, physiopathology
Animals
*Antibody Formation: DE, drug effects
Capillary Permeability: DE, drug effects
Capillary Permeability: PH, physiology
Cyclooxygenase Inhibitors: PD, pharmacology
*Diet
Dinitrophenols: IM, immunology
Enzyme-Linked Immunosorbent Assay
Fatty Acids: BL, blood
Histamine Release: DE, drug effects
Immunoglobulin E: AN, analysis
Indomethacin: PD, pharmacology
***Linoleic Acids: PD, pharmacology**
Lipids: BL, blood
Mice
Mice, Inbred C3H
***alpha-Linolenic Acid: PD, pharmacology**

RN 37341-29-0 (Immunoglobulin E); **463-40-1 (alpha-Linolenic Acid)**; 53-86-1 (Indomethacin)

CN 0 (Cyclooxygenase Inhibitors); 0 (Dinitrophenols); 0 (**Fatty Acids**); 0 (**Linoleic Acids**); 0 (**Lipids**)

L109 ANSWER 14 OF 30 MEDLINE on STN

AN 96131218 MEDLINE

DN PubMed ID: 8523633

TI Effect of different combinations of dietary additives on bacterial translocation and survival in gut-derived **sepsis.**

AU Gennari R; Alexander J W; Eaves-Pyles T

CS University of Cincinnati Medical Center, Department of Surgery, OH 45267-0558, USA.

NC AI-12936 (NIAID)

SO JPEN. Journal of parenteral and enteral nutrition, **(1995 Jul-Aug)** 19 (4) 319-25.
Journal code: 7804134. ISSN: 0148-6071.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199601

ED Entered STN: 19960219
Last Updated on STN: 19980206
Entered Medline: 19960125

AB BACKGROUND: Dietary arginine, glutamine, and **fish oil** each have been shown to improve resistance to infection. The purpose of this study was to assess the potential benefit of different combinations and amounts of these components on bacterial translocation and related mortality during gut-derived **sepsis.** METHODS: Balb/c mice were fed for 10 days with an AIN-76A diet supplemented with different combinations and percentages of arginine, glutamine, glycine, **fish oil**, and **medium-chain triglycerides.**

Controls were fed a complete AIN-76A diet or chow. After 10 days of feeding, all animals were transfused. On day 15, the animals were gavaged with 10(10) 111In-radiolabeled or unlabeled Escherichia coli and given a 30% burn injury. Animals gavaged with unlabeled bacteria were observed for survival (n = 317). Groups that showed the best survival as well as control groups were gavaged with labeled bacteria and killed 4 hours postburn (n = 60) for harvest of mesenteric lymph nodes, liver and spleen. RESULTS: Mice fed diets enriched with 5% **fish oil** + 2% arginine, 2% arginine + 2% glutamine, or 5% **fish oil** + 2% glutamine had higher survival than control groups. The animals fed **fish oil**+glutamine had significantly reduced translocation to the liver and spleen. Animals fed arginine+glutamine had an enhanced ability to kill translocated organisms in the liver compared with other groups. **Fish oil**+arginine improved both barrier function and microbial killing. CONCLUSIONS: Feeding with arginine+glutamine, **fish oil**+arginine, or **fish oil**+glutamine supplemented diets positively affects the outcome in a gut-derived **sepsis** model.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Animals
Arginine: PD, pharmacology
*Bacterial Translocation: DE, drug effects
Digestive System: MI, microbiology
Escherichia coli: DE, drug effects
*Escherichia coli: PH, physiology
*Escherichia coli Infections: PP, physiopathology
Fish Oils: PD, pharmacology
*Food Additives: PD, pharmacology
Food, Fortified
Glutamine: PD, pharmacology
Glycine: PD, pharmacology
Liver: MI, microbiology
Mice
Mice, Inbred BALB C
Random Allocation
Spleen: MI, microbiology
Triglycerides: PD, pharmacology

RN 56-40-6 (Glycine); 56-85-9 (Glutamine); 74-79-3 (Arginine)

CN 0 (**Fish Oils**); 0 (Food Additives); 0 (**Triglycerides**)

L109 ANSWER 15 OF 30 MEDLINE on STN

AN 96044639 MEDLINE

DN PubMed ID: 7552772

TI Effects of dietary alpha- and **gamma-linolenic acids** on liver **fatty acids**, **lipid** metabolism, and survival in **sepsis.**

AU Larsson-Backstrom C; Lindmark L; Svensson L

CS Department of Experimental Biology, Pharmacia AB, Hospital Care, Stockholm, Sweden.

SO Shock (Augusta, Ga.), **(1995 Jul)** 4 (1) 11-20.
Journal code: 9421564. ISSN: 1073-2322.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199511

ED Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951113

AB The effects of dietary treatment for 3 weeks with soybean oil, linseed- and safflower oil (high **alpha-linolenic acid**

, ALA), or borage oil (high **gamma-linolenic acid**, GLA) on the liver **fatty acid** profile and **lipid** metabolism in fed rats, in normal fasted rats, and **septic** fasted rats, and on survival from **sepsis**, were studied. The results were the following: 1) Dietary ALA increased incorporation of alpha-**linolenic** (18:3w3), **eicosapentaenoic** (20:5w3), and **docosapentaenoic** (22:5w3) acids in neutral **lipids** and phospholipids, and **docosahexaenoic** (22:6w3), dihomo-gamma-**linolenic** (20:3w6), **arachidonic** (20:4w6), stearic (18:0), oleic (18:1w9), and **linoleic** (18:2w6) acids in phospholipids in the livers of fed, fasted, and **septic** fasted rats. Dietary GLA increased all w6 **fatty acids** except 18:2w6, and reduced all w3 **fatty acids** in neutral **lipids** and phospholipids. 2) Dietary ALA increased liver phospholipid content in fasted as well as in **septic** fasted rats and was more potent than GLA in lowering serum cholesterol and liver neutral **lipids.** 3) Dietary ALA counteracted **sepsis**-related changes in liver weight, platelet count, body temperature, prekallikrein, serum glucose, beta-hydroxybutyrate, and free **fatty acids.** 4) Dietary GLA reduced survival from **sepsis.** The results suggest a role for w3 **fatty acids** to balance w6 **fatty acids** in the **septic** state.

CT Check Tags: Comparative Study; Male
Animals
Cholesterol: BL, blood
Disease Models, Animal
Fatty Acids: AN, analysis
*Liver: DE, drug effects
Liver: ME, metabolism
Phospholipids: CH, chemistry
Phospholipids: ME, metabolism
Rats
Rats, Sprague-Dawley
Sepsis: DH, diet therapy
***Sepsis: ME, metabolism**
Triglycerides: BL, blood
***alpha-Linolenic Acid: PD, pharmacology**
***gamma-Linolenic Acid: PD, pharmacology**

RN **463-40-1 (alpha-Linolenic Acid); 506-26-3 (gamma-Linolenic Acid)**; 57-88-5 (Cholesterol)

CN 0 (**Fatty Acids**); 0 (Phospholipids); 0 (**Triglycerides**)

L109 ANSWER 16 OF 30 MEDLINE on STN

AN 96026991 MEDLINE

DN PubMed ID: 7475985

TI Enteral feeding a structured **lipid** emulsion containing **fish oil** prevents the fatty liver of **sepsis.**

AU Lanza-Jacoby S; Phetteplace H; Tripp R

CS Department of Surgery, Jefferson Medical College, Philadelphia, Pennsylvania 19107, USA.

NC GM31828 (NIGMS)

SO Lipids, **(1995 Aug)** 30 (8) 707-12.
Journal code: 0060450. ISSN: 0024-4201.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199512

ED Entered STN: 19960124
Last Updated on STN: 19960124
Entered Medline: 19951215

AB **Fish oils** (FO) have been shown to reduce plasma **triglycerides** (TG). In this study we evaluated whether enteral feeding with a structured **lipid** emulsion (SLE) containing FO and **medium-chain triglycerides** (MCT) would prevent the **hypertriglyceridemia** and fatty infiltration of the liver that develops during **sepsis.** For five days, male Lewis rats (275-300 g) were fed intragastrically a nutritionally complete diet containing a SLE or a similar diet with a soybean oil emulsion (SOE) in place of the SLE. On the fifth day, **sepsis** was induced by intravenously injecting 8 x 10(7) live Escherichia coli colonies/100 g b.w.; 24 h later the control SLE, **septic** SLE, control SOE, and **septic** SOE rats were sacrificed. Diet, but not treatment, had a significant effect on serum TG and free **fatty acids** (FFA). Feeding the SLE reduced the plasma FFA of the control and **septic** rats by more than 50% in comparison to both control and **septic** rats fed the SOE. Soleus muscle activity of lipoprotein lipase from the **septic** SLE rats was 44% higher than the control SLE rats. Soleus muscle from the **septic** SLE rats also had a twofold greater activity of lipoprotein lipase than the **septic** SOE rats. TG did not accumulate in the livers of the **septic** rats fed SLE when compared to the control SLE rats and the rats fed the SOE. Livers from the **septic** rats fed the SLE had a third of the TG that were present in the livers from the **septic** rats fed the SOE.(ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Male; Support, U.S. Gov't, P.H.S.
Animals
Cholesterol: BL, blood
Emulsions
*Enteral Nutrition
***Fatty Acids: AD, administration & dosage**
Fatty Acids, Nonesterified: BL, blood
Fatty Liver: ET, etiology
*Fatty Liver: PC, prevention & control
***Fish Oils: AD, administration & dosage**
Fish Oils: TU, therapeutic use
Hypertriglyceridemia: PC, prevention & control
Liver: ME, metabolism
Rats
Rats, Inbred Lew
***Sepsis: CO, complications**
Soybean Oil: AD, administration & dosage
***Triglycerides: AD, administration & dosage**
Triglycerides: BL, blood
Triglycerides: ME, metabolism

RN 57-88-5 (Cholesterol); 8001-22-7 (Soybean Oil)

CN 0 (Emulsions); 0 (**Fatty Acids**); 0 (**Fatty Acids**, Nonesterified); 0 (**Fish Oils**); 0 (**Triglycerides**)

L109 ANSWER 17 OF 30 MEDLINE on STN

AN 95178881 MEDLINE

DN PubMed ID: 7873917

TI Chemically defined structured **lipids** with **omega-3 fatty acids** maintain splanchnic blood flow in a low-dose continuous endotoxin model.

AU Pscheidl E; Reisch S; Rugheimer E

CS Institut fur Anaesthesiologie, Universitat Erlangen-Nurnberg, FRG.

SO Infusionstherapie und Transfusionsmedizin, **(1994 Dec)** 21 (6) 380-7.
Journal code: 9209406. ISSN: 1019-8466.

CY Switzerland

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals
EM 199504
ED Entered STN: 19950419
Last Updated on STN: 19950419
Entered Medline: 19950406
AB BACKGROUND: Disturbances of microcirculation and accompanying alterations of oxygen supply are central pathophysiological events in trauma and **sepsis.** There is evidence that **omega-3 fatty acids** can modulate prostaglandin formation and thereby regional blood flow. The aim of the study was to determine the effects of chemically defined structured **lipids** (SL) with **omega-3 fatty acids** in position sn-2 (MFM) compared to SL with **omega-6 fatty acids** in position sn-2 (MLM) on cardiac output (CO) and splanchnic blood flow in a low-dose endotoxin (E, 1 mg.kgBW-1.day-1) rat model. MATERIALS AND METHODS: 24 male Sprague Dawley rats, divided in 4 groups (**n = 6**; MLM, MLM+E, MFM, MFM+E) received for 48 h a total parenteral nutrition. CO and regional blood flow were measured with 85strontium-labelled microspheres (16.5 +/- 0.1 microns). RESULTS: There was a slight rise in CO in the E groups compared to the control groups. Application of E resulted in a marked decrease of intestinal perfusion in the MLM-fed animals, whereas the MFM-fed animals showed only a minimal reduction. This decrease of portal blood flow to the liver was accompanied by an elevation of arterial blood flow to the liver. This compensatory increase in arterial liver blood flow was more pronounced in the MFM-fed animals, resulting in a total liver blood flow which was not different from the control group. CONCLUSIONS: The results of this study implicate that 48 h of intravenous feeding with chemically defined SL with an **omega-3 fatty acid** in position sn-2 can significantly influence splanchnic bed perfusion in a low-dose endotoxin rat model. The better splanchnic perfusion may be mediated by a shift in prostaglandin production.
CT Check Tags: Comparative Study; Male
Animals
Digestive System: BS, blood supply
*Endotoxins: BL, blood
Energy Metabolism: PH, physiology
*Escherichia coli
*Fat Emulsions, Intravenous
***Fatty Acids, Omega-3: PD, pharmacology**
Fatty Acids, Omega-6
Fatty Acids, Unsaturated: PD, pharmacology
Hemodynamic Processes: PH, physiology
Kidney: BS, blood supply
Liver: BS, blood supply
Liver Circulation: PH, physiology
Lung: BS, blood supply
Muscles: BS, blood supply
*Parenteral Nutrition, Total
Rats
Rats, Sprague-Dawley
Regional Blood Flow: PH, physiology
***Shock, Septic: PP, physiopathology**
CN 0 (Endotoxins); 0 (Fat Emulsions, Intravenous); 0 (**Fatty Acids, Omega-3**); 0 (**Fatty Acids, Omega-6**); 0 (**Fatty Acids**, Unsaturated)

L109 ANSWER 18 OF 30 MEDLINE on STN
AN 95006769 MEDLINE
DN PubMed ID: 7922448
TI **Omega-3** polyunsaturated **fatty acids** : benefit or harm during **sepsis**?.

AU Peck M D
CS University of Miami/Jackson Memorial Burn Center, University of Miami School of Medicine, FL 33101.
SO New horizons (Baltimore, Md.), **(1994 May)** 2 (2) 230-6. Ref: 68
Journal code: 9416195. ISSN: 1063-7389.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941025
AB **omega-3** polyunsaturated **fatty acids** (PUFAs) are potent modulators of the immune response. Their inclusion in enteral diets may benefit surgical patients recovering from injury or infection. Caution should be used when supplementing **omega-3** PUFAs, particularly when **fish oil** is used as the source. The long-chain, highly polyunsaturated **fatty acids** in **fish oil** are prone to autoxidation, and can potentially damage cells by forming free radicals. In addition, **fish oil** may impair the hemostatic response by inhibiting platelet aggregation. Finally, the biochemical and biological end points for the use of **omega-3** PUFAs have not been clearly established. Thus, although **omega-3** PUFAs offer tremendous potential as immunomodulators, they also offer potential toxicities, and we must employ them in our clinical practice with this understanding.
CT Check Tags: Human
Biological Response Modifiers: IM, immunology
Biological Response Modifiers: PD, pharmacology
*Biological Response Modifiers: TU, therapeutic use
*Eicosanoids: BI, biosynthesis
*Enteral Nutrition: AE, adverse effects
Enteral Nutrition: MT, methods
Fatty Acids, Omega-3: IM, immunology
Fatty Acids, Omega-3: PD, pharmacology
***Fatty Acids, Omega-3: TU, therapeutic use**
Fish Oils: IM, immunology
Fish Oils: PD, pharmacology
Fish Oils: TU, therapeutic use
Free Radicals
Infection: IM, immunology
Infection: ME, metabolism
*Infection: TH, therapy
Lipid Peroxidation
Platelet Aggregation: DE, drug effects
CN 0 (Biological Response Modifiers); 0 (Eicosanoids); 0 (**Fatty Acids, Omega-3**); 0 (**Fish Oils**); 0 (Free Radicals)

L109 ANSWER 19 OF 30 MEDLINE on STN
AN 94376160 MEDLINE
DN PubMed ID: 7916376
TI A high alpha-linolenate diet suppresses antigen-induced immunoglobulin E response and anaphylactic **shock** in mice.
AU Watanabe S; Sakai N; Yasui Y; Kimura Y; Kobayashi T; Mizutani T; Okuyama H
CS Department of Biological Chemistry, Faculty of Pharmaceutical Sciences, Nagoya City University, Japan.
SO Journal of nutrition, **(1994 Sep)** 124 (9) 1566-73.
Journal code: 0404243. ISSN: 0022-3166.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 19941031
Last Updated on STN: 19980206
Entered Medline: 19941020
AB Mice were fed for 2 mo diets having ratios of alpha-linolenate [18:3 (**n-3**)] to linoleate [18:2(**n-6**)] of < 0.01, 0.36, 1.0 and 3.9. Proportions of safflower seed oil and perilla seed oil were adjusted to obtain these ratios. The dietary alpha-linolenate to linoleate balance was reflected in the proportion of (**n-3**) and (**n-6**) highly unsaturated **fatty acids** with 20- and 22-carbon chains in spleen phospholipids, but the ratio did not affect the proportion of T lymphocyte subsets expressing CD4 and CD8 antigens in splenic leukocytes. The immunoglobulin (Ig) G and IgM responses against sheep red blood cells when estimated as plaque-forming cells present in spleen, were not affected significantly by the diets. However, the serum hemagglutinin titer was slightly but significantly higher in the high alpha-linolenate diet group [18:3(**n-3**)/18:2(**n-6**) = 3.9] than in the dietary group with 18:3(**n-3**) to 18:2(**n-6**) ratios of 0.36 and < 0.01. In contrast, the IgE antibody response against egg albumin, as well as the mortality from anaphylactic **shock** induced by a second challenge with antigen, was significantly lower in the high alpha-linolenate diet group [18:3(**n-3**)/18:2(**n-6**) = 3.9] than in the high linoleate diet [18:3(**n-3**)/18:2(**n-6**) < 0.01] group. These results, together with the reported suppressive effects of a high alpha-linolenate diet on the formation of **lipid**-derived allergic mediators, support the hypothesis that raising the (**n-3**) to (**n-6**) ratios of diets would be effective in reducing the severity of immediate-type allergic hypersensitivity.
CT Check Tags: Male; Support, Non-U.S. Gov't
*Anaphylaxis: PC, prevention & control
Animals
*Dietary Fats, Unsaturated: AD, administration & dosage
Fatty Acids: AN, analysis
Hemagglutinins: AN, analysis
*Hypersensitivity, Immediate: PC, prevention & control
*Immunoglobulin E: BI, biosynthesis
Immunoglobulin G: BI, biosynthesis
Immunoglobulin M: BI, biosynthesis
Linoleic Acid
Linoleic Acids: AD, administration & dosage
Mice
Mice, Inbred C3H
Mice, Inbred ICR
Ovalbumin: IM, immunology
Phospholipids: CH, chemistry
Spleen: CH, chemistry
Spleen: CY, cytology
T-Lymphocyte Subsets
***alpha-Linolenic Acid: AD, administration & dosage**
RN **2197-37-7 (Linoleic Acid)**; 37341-29-0 (Immunoglobulin E); **463-40-1 (alpha-Linolenic Acid)**; 9006-59-1 (Ovalbumin)
CN 0 (Dietary Fats, Unsaturated); 0 (**Fatty Acids**); 0 (Hemagglutinins); 0 (Immunoglobulin G); 0 (Immunoglobulin M); 0 (**Linoleic Acids**); 0 (Phospholipids)

L109 ANSWER 20 OF 30 MEDLINE on STN

AN 94318785 MEDLINE
DN PubMed ID: 8043713
TI [Fats in parenteral nutrition].
Fett in der parenteralen Ernahrungstherapie.
AU Schricker T; Georgieff M
CS Universitatsklinik fur Anasthesiologie, Klinikum der Universitat Ulm.
SO Anasthesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie : AINS, **(1994 May)** 29 (3) 137-45. Ref: 45
Journal code: 9109478. ISSN: 0939-2661.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 199408
ED Entered STN: 19940909
Last Updated on STN: 19940909
Entered Medline: 19940831
AB This paper aims at presenting the properties and actions of individual **lipids**, as determined via various animal experiments, on the organ systems of the area of the splanchnicus and the lung that are of particular relevance for the intensive-care physician, and to derive possible therapeutic options form these data. Although so far no data are available on newly introduced kinds of fat such as **fish oil** used in parenteral feeding of critically ill patients, it will be the aim of future therapeutic concepts to arrive at optimal combinations for the individual patient of the pharmacological, energetic and essential properties of the individual classes of **fatty acids.** Structured **lipids** may possibly be suitable for this task in an ideal manner, the glycerol molecule of which is esterified with **linoleic acid** as an essential constituent of food, a **medium-chain fatty acid** as energy carrier, and an **omega-3 fatty acid** as immunomodulator. The question as to whether such mixtures can eventually be administered in a not too distant future to parenterally fed patients after trauma and during **septic** condition for their benefit, can be answered only by means of clinical studies that will have to be conducted in years to come.
CT Check Tags: Human
Animals
*Critical Care
Energy Metabolism: PH, physiology
English Abstract
*Fat Emulsions, Intravenous: AD, administration & dosage
Fat Emulsions, Intravenous: CL, classification
Fat Emulsions, Intravenous: ME, metabolism
Nutritional Requirements
*Parenteral Nutrition, Total
CN 0 (Fat Emulsions, Intravenous)

L109 ANSWER 21 OF 30 MEDLINE on STN
AN 93243304 MEDLINE
DN PubMed ID: 8480680
TI Rapid incorporation of fish or olive oil **fatty acids** into rat hepatic sinusoidal cell phospholipids after continuous enteral feeding during endotoxemia.
AU Palombo J D; Bistrian B R; Fechner K D; Blackburn G L; Forse R A
CS Department of Surgery, New England Deaconess Hospital, Harvard Medical School, Boston, MA 02215.
NC DK31933 (NIDDK)
SO American journal of clinical nutrition, **(1993 May)** 57 (5) 643-9.
Journal code: 0376027. ISSN: 0002-9165.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199305
ED Entered STN: 19930611
Last Updated on STN: 19930611
Entered Medline: 19930527
AB Therapeutic modalities that downregulate macrophage and endothelial production of eicosanoid mediators by displacing membrane **arachidonic acid** (20:4 **omega 6**) may benefit patients at increased risk of **septic** complications. The objective of this study in rats was to assess the incorporation of fish or olive oil **fatty acids** into hepatic Kupffer and endothelial (K&E) cell phospholipids after 4 d of continuous enteral feeding during endotoxemia. Either endotoxin (ETX) (0.5-1 mg-1.day-1) or vehicle was infused intravenously during the last 72 h. Dietary fish and olive oil **fatty acids** were rapidly incorporated into both K&E and plasma phospholipids irrespective of ETX cotreatment. Rats infused with the **fish oil**-enriched diet had a significantly lower relative percent of both K&E **linoleic acid** (18:2 **omega 6**) and 20:4 **omega 6**, whereas rats infused with the olive oil-enriched diet only had a lower relative percent of 18:2 **omega 6** compared with control rats receiving corn oil. Provision of specific dietary **lipids** by continuous enteral infusion may prove efficacious for the rapid modulation of hepatic sinusoidal cell membrane **fatty acids** under either normal or endotoxemic conditions.
CT Check Tags: Male; Support, U.S. Gov't, P.H.S.
Animals
Dietary Fats: ME, metabolism
Endothelium: CY, cytology
Endothelium: DE, drug effects
Endothelium: ME, metabolism
Endotoxins
Enteral Nutrition
Escherichia coli
***Fatty Acids: ME, metabolism**
Fatty Acids: PD, pharmacology
***Fish Oils: ME, metabolism**
Fish Oils: PD, pharmacology
Kupffer Cells: DE, drug effects
Kupffer Cells: ME, metabolism
Liver: CY, cytology
Liver: DE, drug effects
*Liver: ME, metabolism
*Phospholipids: ME, metabolism
*Plant Oils: ME, metabolism
Plant Oils: PD, pharmacology
Rats
Rats, Sprague-Dawley
Toxemia: DH, diet therapy
*Toxemia: ME, metabolism
RN 8001-25-0 (olive oil)
CN 0 (Dietary Fats); 0 (Endotoxins); 0 (**Fatty Acids**); 0 (**Fish Oils**); 0 (Phospholipids); 0 (Plant Oils)

L109 ANSWER 22 OF 30 MEDLINE on STN
AN 92349634 MEDLINE
DN PubMed ID: 1640634
TI Adaptation to a **fish oil** diet before inducing **sepsis** in rats prevents fatty infiltration of the liver.
AU Lanza-Jacoby S; Smythe C; Phetteplace H; Tabares A

CS Department of Surgery, Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA 19107.
NC GM3128 (NIGMS)
SO JPEN. Journal of parenteral and enteral nutrition, **(1992 Jul-Aug)** 16 (4) 353-8.
Journal code: 7804134. ISSN: 0148-6071.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199209
ED Entered STN: 19920911
Last Updated on STN: 19970203
Entered Medline: 19920903
AB **Hypertriglyceridemia** and fatty liver are common **lipid** abnormalities associated with Gram-negative **sepsis.** **Fish oils** have been shown to have beneficial effects in reducing plasma **triglycerides** (TG). This study was designed to investigate whether **fish oils** would prevent the elevation of plasma TG and the accumulation of liver **lipids** during **sepsis.** One group of rats was fed a 10% menhaden oil diet and the other group was fed a 10% corn oil diet for 14 days. On the 14th day, **sepsis** was induced by injecting the rats with 8 x 10(7) live Escherichia coli colonies/100 g of body weight and the rats were fasted for 22 hours. The liver composition of total **lipids** and TG in the **septic** rats prefed the **fish oil** was lower than in the **septic** rats prefed the corn oil. In the rats adapted to the corn oil diet, **lipids** accumulated in the livers of the **septic** rats in comparison with the control rats. Hepatocytes isolated from the **septic** rats adapted to the corn oil diet showed an increased esterification of [1-14C]palmitate into TG and phospholipids than hepatocytes from the control rats. Feeding the **fish oil** diet instead of the corn oil diet before inducing **sepsis** reduced TG, cholesterol, and phospholipid synthesis by 58%, 79%, and 71%, respectively. The rise in TG synthesis in the **septic** rats prefed the corn oil diet was associated with an 89% increase in the activity of phosphatidate phosphohydrolase. There was no significant difference in the activities of glycerol-3-phosphate acyltransferase and phosphatidate phosphohydrolase between control and **septic** rats.(ABSTRACT TRUNCATED AT 250 WORDS)
CT Check Tags: Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Animals
*Dietary Fats, Unsaturated: TU, therapeutic use
Escherichia coli Infections
Esterification
Fatty Liver: ET, etiology
Fatty Liver: ME, metabolism
*Fatty Liver: PC, prevention & control
***Fish Oils: TU, therapeutic use**
Lipids: BL, blood
Lipoprotein Lipase: ME, metabolism
Liver: ME, metabolism
Palmitic Acid
Palmitic Acids: ME, metabolism
Phospholipids: ME, metabolism
Rats
Rats, Inbred Lew
***Septicemia: CO, complications**
Septicemia: ME, metabolism
Triglycerides: BL, blood
Triglycerides: ME, metabolism
RN 57-10-3 (Palmitic Acid); 8002-50-4 (Menhaden oil)
CN 0 (Dietary Fats, Unsaturated); 0 (**Fish Oils**); 0 (

Lipids); 0 (Palmitic Acids); 0 (Phospholipids); 0 (**Triglycerides**); EC 3.1.1.34 (Lipoprotein Lipase)

L109 ANSWER 23 OF 30 MEDLINE on STN
AN 92318760 MEDLINE
DN PubMed ID: 1619987
TI Influence of **omega-3 fatty acids** on splanchnic blood flow and lactate metabolism in an endotoxemic rat model.
AU Pscheidl E M; Wan J M; Blackburn G L; Bistrian B R; Istfan N W
CS Nutrition-Metabolism Laboratory, New England Deaconess Hospital, Harvard Medical School, Boston, MA 02215.
NC DK 40252 (NIDDK)
DK 40492 (NIDDK)
GM 34074 (NIGMS)
SO Metabolism: clinical and experimental, **(1992 Jul)** 41 (7) 698-705.
Journal code: 0375267. ISSN: 0026-0495.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199207
ED Entered STN: 19920815
Last Updated on STN: 19970203
Entered Medline: 19920731
AB Alteration in regional blood flow is important in the pathogenesis of organ failure during endotoxemia and **sepsis.** In particular, intestinal ischemia is thought to enhance the translocation of bacteria into the systemic circulation. We used radioactive microspheres to measure the influence of two intravenous (IV) dietary fats (vegetable oil containing high levels of **omega-6 fatty acids**, and **fish oil** containing high levels of **omega-3 fatty acids**) on regional blood flow during low-dose Escherichia coli endotoxin infusion (0.1 mg/100 g body weight [BW]) in a rat model. Despite absence of changes in the cardiac output, blood flow rates to the small and large intestines, stomach, and pancreas, and also to the skin and skeletal muscle were significantly reduced after 18 hours of endotoxin infusion in the rats fed standard vegetable oil. Short-term IV feeding during a period of 40 hours with an isonitrogenous, isocaloric nutrient solution containing **fish oil** as the only **lipid** source normalized intestinal perfusion and increased blood flow to the liver and spleen. Low-dose endotoxin infusion also resulted in significant increases in glucose, lactate, and pyruvate concentrations. In comparison to standard vegetable fat emulsion, **fish oil** significantly reduced these parameters. A second experiment was conducted to measure lactate kinetics. Based on the dilution of U-14C-lactate, **fish oil** feeding was associated with higher lactate clearance than standard vegetable oil feeding during the endotoxin infusion. We conclude that short-term IV feeding with **fish oil** improves intestinal perfusion and portal blood flow, improves glucose tolerance, and increases lactate clearance in a low-dose endotoxin rat model.
CT Check Tags: Male; Support, U.S. Gov't, P.H.S.
Animals
Cardiac Output: DE, drug effects
*Endotoxins: TO, toxicity
***Fatty Acids, Omega-3: PD, pharmacology**
Fish Oils: PD, pharmacology
Glucose: ME, metabolism
*Lactates: ME, metabolism
Lactic Acid
Rats

Rats, Inbred Strains
*Splanchnic Circulation: DE, drug effects
Thromboxane A2: PH, physiology
*Toxemia: PP, physiopathology
RN 50-21-5 (Lactic Acid); 50-99-7 (Glucose); 57576-52-0 (Thromboxane A2)
CN 0 (Endotoxins); 0 (**Fatty Acids, Omega-3**); 0 (**Fish Oils**); 0 (Lactates)

L109 ANSWER 24 OF 30 MEDLINE on STN
AN 92048543 MEDLINE
DN PubMed ID: 1943744
TI Long-term feeding with structured **lipid** composed of **medium-chain** and **N-3 fatty acids** ameliorates endotoxic **shock** in guinea pigs.
AU Teo T C; Selleck K M; Wan J M; Pomposelli J J; Babayan V K; Blackburn G L; Bistrian B R
CS Department of Surgery, Aberdeen Royal Infirmary, Foresterhill, Scotland.
NC DK 40252 (NIDDK)
DK 41128 (NIDDK)
SO Metabolism: clinical and experimental, **(1991 Nov)** 40 (11) 1152-9.
Journal code: 0375267. ISSN: 0026-0495.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199112
ED Entered STN: 19920124
Last Updated on STN: 19970203
Entered Medline: 19911212
AB The metabolic and physiologic responses to 7-hour endotoxin infusion (5.0 mg/kg h) were evaluated in guinea pigs following 6 weeks of dietary enrichment with diets containing either chemically structured **lipid** (SL) composed of **medium-chain triglycerides** (MCT) and long-chain **triglycerides** (LCT) in the form of **N-3** polyunsaturated **fatty acids** (PUFAs), or safflower oil (SO), which is high in **N-6 fatty acids.** Plasma phospholipid **fatty acid** profiles, arterial blood pH, PCO2, PO2, HCO3, lactate, blood pressure, oxygen consumption, and energy expenditure were examined. Plasma phospholipid **fatty acids** profiles reflected dietary intake with SL-fed animals demonstrating a significantly higher **N-3** to **N-6 fatty acid** ratio compared with SO-fed animals. SL-fed animals responded to endotoxemia with a mild metabolic acidosis with respiratory compensation, which was associated with moderate lactatemia (3 mmol/L). SO-fed animals developed a severe metabolic acidosis with acidemia and respiratory compensation, which was associated with hyperlactatemia (8 mmol/L, P less than .05 v SL). No differences were observed in blood pressure, oxygen consumption, energy expenditure, or respiratory quotient during endotoxemia between dietary groups compared with controls. We conclude that diets enriched with structured **lipid** composed of **medium-chain** and **N-3 fatty acids** can attenuate the sequelae of endotoxemia.
CT Check Tags: Male; Support, U.S. Gov't, P.H.S.
Animals
Blood Pressure
Calorimetry, Indirect
*Dietary Fats: PD, pharmacology
Fatty Acids: BL, blood
***Fatty Acids: PD, pharmacology**
Guinea Pigs
Lactates: BL, blood

Lactic Acid
Lipids: CH, chemistry
***Lipids: PD, pharmacology**
Phospholipids: BL, blood
Shock, Septic: ME, metabolism
***Shock, Septic: PP, physiopathology**
Time Factors
RN 50-21-5 (Lactic Acid)
CN 0 (Dietary Fats); 0 (**Fatty Acids**); 0 (Lactates); 0 (**Lipids**); 0 (Phospholipids)

L109 ANSWER 25 OF 30 MEDLINE on STN
AN 91318319 MEDLINE
DN PubMed ID: 1861166
TI High dietary **linoleic acid** affects the **fatty acid** compositions of individual phospholipids from tissues of Atlantic salmon (Salmo salar): association with stress susceptibility and cardiac lesion.
AU Bell J G; McVicar A H; Park M T; Sargent J R
CS Unit of Aquatic Biochemistry, School of Natural Sciences, University of Stirling, Scotland.
SO Journal of nutrition, **(1991 Aug)** 121 (8) 1163-72.
Journal code: 0404243. ISSN: 0022-3166.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199108
ED Entered STN: 19910922
Last Updated on STN: 19980206
Entered Medline: 19910830
AB For 16 wk Atlantic salmon (Salmo salar) post-smolts were fed practical-type diets that contained either **fish oil** (FO) or sunflower oil (SO) as the **lipid** component. Both diets contained adequate (**n-3**) polyunsaturated **fatty acids** (PUFA). All the phospholipids of heart and liver from SO-fed fish had increased levels of 18:2(**n-6**), 20:2(**n-6**) and 20:3(**n-6**); phosphatidyl choline (PC) and phosphatidyl ethanolamine (PE) also had increased 20:4(**n-6**). There was a general decrease in 20:5(**n-3**) in the phospholipids, reflected in an increase in the 20:4(**n-6**)/20:5(**n-3**) ratio, especially in PC and PE. The **fatty acid** compositions of phospholipids from brain and retina were much less affected by dietary linoleate than those of heart and liver. Fish fed SO developed severe heart lesions that caused thinning of the ventricular wall and muscle necrosis. The fish fed SO also were susceptible to a transportation-induced **shock** syndrome that caused 30% mortality. These results establish that a diet with a low (**n-3**)/(**n-6**) ratio can cause changes in **fatty acid** metabolism that are deleterious to the health of salmonid fish, especially when subjected to stress.
CT Animals
Brain: DE, drug effects
Brain: ME, metabolism
Dietary Fats, Unsaturated: AD, administration & dosage
*Dietary Fats, Unsaturated: PD, pharmacology
***Fatty Acids: ME, metabolism**
Fish Oils: AD, administration & dosage
Fish Oils: PD, pharmacology
Heart: DE, drug effects
Heart Diseases: ET, etiology
Heart Diseases: PA, pathology

*Heart Diseases: VE, veterinary
Linoleic Acid
Linoleic Acids: AD, administration & dosage
Linoleic Acids: AE, adverse effects
***Linoleic Acids: PD, pharmacology**
Liver: DE, drug effects
Liver: ME, metabolism
Myocardium: ME, metabolism
Myocardium: PA, pathology
Necrosis
Phosphatidylcholines: ME, metabolism
Phosphatidylethanolamines: ME, metabolism
*Phospholipids: ME, metabolism
Plant Oils: AD, administration & dosage
Plant Oils: PD, pharmacology
Retina: DE, drug effects
Retina: ME, metabolism
*Salmon
Stress: ET, etiology
*Stress: VE, veterinary
RN **2197-37-7 (Linoleic Acid)**; 8001-21-6 (sunflower seed oil)
CN 0 (Dietary Fats, Unsaturated); 0 (**Fatty Acids**); 0 (**Fish Oils**); 0 (**Linoleic Acids**); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phospholipids); 0 (Plant Oils)

L109 ANSWER 26 OF 30 MEDLINE on STN
AN 91269439 MEDLINE
DN PubMed ID: 1904949
TI Effects of a **fish oil** diet on pigs' cardiopulmonary response to bacteremia.
AU Murray M J; Svingen B A; Holman R T; Yaksh T L
CS Critical Care Service, Mayo Clinic, Rochester, MN 55905.
SO JPEN. Journal of parenteral and enteral nutrition, **(1991 Mar-Apr)** 15 (2) 152-8.
Journal code: 7804134. ISSN: 0148-6071.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107
ED Entered STN: 19910811
Last Updated on STN: 19910811
Entered Medline: 19910722
AB Since an **omega 3 fatty acid** (FA) diet may have beneficial effects in inflammatory processes, we tested the hypothesis that the physiologic response to **sepsis** could be modified by altering the eicosanoid precursor pool via an **omega 3** FA diet. Two groups (n = 8) of pigs were prefed for 8 days either an **omega 3** FA or an **omega 6** FA diet (Weaner Pig Feed with either menhaden or corn oil to produce a eucaloric feed with 15% fat) and then injected with live Escherichia coli. The **omega 3** FA diet increased the concentration of eicosapentainoic acid (EPA, 20:5 **omega 3**) in plasma **lipids**, and increased the ratio of EPA to **arachidonic acid** (AA, 20:4 **omega 6**) in platelets from 1:20 to 1:1 over the 8 days. Following the injection of bacteria, there was a fall in PaO2 and blood pressure that was attenuated (p less than 0.05) by the **omega 3** FA diet. The **omega 3** FA diet, compared to the **omega 6** FA diet, also attenuated the rise in thromboxane B2 (3.0 +/- 1.1 vs 12.9 +/- 5.7 ng/mL) and 6 keto-PGF1 alpha (0.8 +/- 0.5 vs 1.7 +/- 1.1 ng/mL) associated with bacteremia. We conclude that dietary **omega 3** FA

attenuated the physiologic response to **sepsis**, possibly by modifying **arachidonic acid** metabolism.

CT Check Tags: Male
6-Ketoprostaglandin F1 alpha: BL, blood
Animals
Arachidonic Acid
Arachidonic Acids: BL, blood
Blood Platelets: ME, metabolism
Blood Pressure
*Cardiovascular System: PP, physiopathology
*Dietary Fats, Unsaturated: TU, therapeutic use
Eicosapentaenoic Acid: BL, blood
Escherichia coli Infections: CO, complications
*Escherichia coli Infections: PP, physiopathology
***Fish Oils: TU, therapeutic use**
Lipids: BL, blood
*Lung: PP, physiopathology
Norepinephrine: BL, blood
Oxygen: BL, blood
Pulmonary Edema: ET, etiology
Septicemia: CO, complications
Septicemia: DH, diet therapy
***Septicemia: PP, physiopathology**
Swine
Thromboxane B2: BL, blood

RN **1553-41-9 (Eicosapentaenoic Acid)**; **506-32-1 (Arachidonic Acid)**; 51-41-2 (Norepinephrine); 54397-85-2 (Thromboxane B2); 58962-34-8 (6-Ketoprostaglandin F1 alpha); 7782-44-7 (Oxygen)

CN 0 (**Arachidonic Acids**); 0 (Dietary Fats, Unsaturated); 0 (**Fish Oils**); 0 (**Lipids**)

L109 ANSWER 27 OF 30 MEDLINE on STN

AN 91187922 MEDLINE

DN PubMed ID: 2011612

TI Free radical generation, **lipid** peroxidation and essential **fatty acids** in patients with **septicemia.**

AU Prabha P S; Das U N; Ramesh G; Kumar K V; Kamalakar V

CS Department of Medicine, Nizam's Institute of Medical Sciences, Hyderabad, India.

SO Prostaglandins, leukotrienes, and essential fatty acids, **(1991 Jan)** 42 (1) 61-5.
Journal code: 8802730. ISSN: 0952-3278.

CY SCOTLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199105

ED Entered STN: 19910526
Last Updated on STN: 19910526
Entered Medline: 19910506

AB Infections due to gram-negative bacteria and other organisms can lead to **septicemia** and **shock** in some patients. Endotoxins, which cause these pathophysiological events, stimulate macrophages to elaborate tumor necrosis factor and other lymphokines. These lymphokines can augment free radical generation by polymorphonuclear leukocytes, macrophages and other cells, which may ultimately produce respiratory distress syndrome, multiorgan failure and irreversible **shock** seen in **septicemia.** This is supported by our results presented here that there is indeed an increase in free radical generation and **lipid** peroxidation in patients with **septicemia.** In addition, analysis of plasma **lipid** profile in these patients showed that gamma-**linolenic**, dihomogamma-**linolenic** and **arachidonic acids** of **n-6** series and

alpha-**linolenic** and **eicosapentaenoic acids** of the **n-3** series are decreased in their plasma phospholipid fraction. These results suggest that free radicals, **lipid** peroxides, and alteration in essential **fatty acid** metabolism may have a role in the pathogenesis of **septicemia.**

CT Check Tags: Human; Support, Non-U.S. Gov't
Adolescent
Adult
Aged
Animals
Dogs
***Fatty Acids: BL, blood**
Free Radicals
*Gram-Negative Bacteria: ME, metabolism
***Lipid Peroxidation**
Lipid Peroxides: BL, blood
Middle Aged
Neutrophils: DE, drug effects
*Neutrophils: ME, metabolism
Phospholipids: BL, blood
***Septicemia: ME, metabolism**
Septicemia: MI, microbiology
Shock

CN 0 (**Fatty Acids**); 0 (Free Radicals); 0 (**Lipid** Peroxides); 0 (Phospholipids)

L109 ANSWER 28 OF 30 MEDLINE on STN
AN 91128027 MEDLINE
DN PubMed ID: 1992944
TI Diets enriched with **N-3 fatty acids** ameliorate lactic acidosis by improving endotoxin-induced tissue hypoperfusion in guinea pigs.
AU Pomposelli J J; Flores E A; Blackburn G L; Zeisel S H; Bistrian B R
CS Cancer Research Institute, New England Deaconess Hospital, Boston, MA 02215.
NC AM31933 (NIADDK)
GM30632 (NIGMS)
SO Annals of surgery, **(1991 Feb)** 213 (2) 166-76.
Journal code: 0372354. ISSN: 0003-4932.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199103
ED Entered STN: 19910405
Last Updated on STN: 19910405
Entered Medline: 19910314
AB The effect of 6 weeks dietary **lipid** manipulation on the acute physiologic response to 7-hour continuous endotoxin infusion in guinea pigs was examined. One diet was enriched with **N-3 fatty acids**, whereas the other contained **N-6 fatty acids**, primarily **linoleic acid.** Animals fed **N-6 fatty acids** developed significant lactic acidemia, microvascular muscle hypoperfusion, and pulmonary infiltrates in response to endotoxin infusion. **N-3 fatty acid**-fed animals demonstrated improved lactate levels, microvascular muscle perfusion, and lung morphology compared to **N-6 fatty acid**-fed animals after endotoxin infusion. There was no significant change in cardiac output, PaO2, or mean arterial blood pressure at the end of the endotoxin infusion in either group. Pretreatment with indomethacin, or BM 13505, a specific thromboxane A2

receptor blocker, ameliorated the development of metabolic acidosis in **N-6 fatty acid**-fed animals, demonstrating a role for prostanoids in the sequelae of endotoxemia. The ability of dietary pretreatment with **N-3 fatty acids** to influence favorably the physiologic response to endotoxin represents a novel nutrient-metabolic interaction with potential therapeutic implications.

CT Check Tags: Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Acidosis, Lactic: BL, blood
Acidosis, Lactic: ET, etiology
Acidosis, Lactic: TH, therapy
Animals
Dietary Fats: AD, administration & dosage
Dietary Fats: PD, pharmacology
Fatty Acids, Omega-3: AD, administration & dosage
Fatty Acids, Unsaturated: AD, administration & dosage
Fish Oils: AD, administration & dosage
Guinea Pigs
*Hemodynamic Processes: DE, drug effects
Indomethacin: PD, pharmacology
Lung: PA, pathology
Muscles: BS, blood supply
Phenylacetates: AD, administration & dosage
Regional Blood Flow: DE, drug effects
Safflower Oil: AD, administration & dosage
Shock, Septic: CO, complications
Shock, Septic: PA, pathology
***Shock, Septic: PP, physiopathology**
Skin: BS, blood supply
Sulfonamides: AD, administration & dosage
Thromboxanes: AI, antagonists & inhibitors

RN 105218-03-9 (daltroban); 53-86-1 (Indomethacin); 8001-23-8 (Safflower Oil)

CN 0 (Dietary Fats); 0 (**Fatty Acids, Omega-3**); 0 (**Fatty Acids**, Unsaturated); 0 (**Fish Oils**); 0 (Phenylacetates); 0 (Sulfonamides); 0 (Thromboxanes)

L109 ANSWER 29 OF 30 MEDLINE on STN

AN 89237146 MEDLINE

DN PubMed ID: 2541281

TI Modulation of Kupffer cell membrane phospholipid function by **n-3** polyunsaturated **fatty acids.**

AU Bankey P E; Billiar T R; Wang W Y; Carlson A; Holman R T; Cerra F B

CS Department of Surgery, University of Minnesota, Minneapolis.

SO Journal of surgical research, **(1989 May)** 46 (5) 439-44.
Journal code: 0376340. ISSN: 0022-4804.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198906

ED Entered STN: 19900306
Last Updated on STN: 19900306
Entered Medline: 19890614

AB Dietary **n-3** polyunsaturated **fatty acids** (PUFAs) have been reported to improve clinical outcome in a number of inflammatory diseases including burns and **sepsis.** One mechanism contributing to the anti-inflammatory effect is the incorporation of **n-3** PUFAs into membrane phospholipids which decreases macrophage eicosanoid production. We hypothesize that an additional mechanism for their effects is an alteration of membrane signal transduction that decreases macrophage responsiveness to inflammatory stimuli. Kupffer cells, the fixed macrophages of the liver, were obtained

from rats pair fed diets for 6 weeks with 15% of calories supplied as menhaden (high **n-3**), corn (control), or safflower (high **n-6**) oils. The effects of the dietary oils on Kupffer cell membrane signal transduction and eicosanoid production were assessed by measuring inositol phospholipid (PI) metabolism, intracellular calcium responses, and prostaglandin E2 (PGE2) production to the inflammatory signals endotoxin (LPS) and platelet activating factor (PAF). The menhaden oil diet resulted in significant incorporation of **n-3** PUFAs into total cellular PUFAs compared to corn and safflower oil. (total **n-3** PUFAs, 28.1% menhaden vs 2.1% corn vs 1.2% safflower, P less than 0.03). This incorporation altered signal transduction of PAF as both PI turnover (65% +/- 10% of corn oil) and calcium response (0.6-fold vs 5.0-fold for corn oil) were significantly reduced in the menhaden oil group. (P less than 0.05) The menhaden oil diet also reduced significantly PGE2 production in response to PAF and LPS (corn, 348 +/- 23 pg/ml; menhaden, 48 +/- 6 pg/ml, P less than 0.01). We conclude that, in addition to modulating eicosanoid production, **n-3** PUFAs can also alter macrophage membrane signal transduction.(ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Male
Animals
Calcium: ME, metabolism
Calcium Channels: ME, metabolism
Corn Oil: ME, metabolism
*Dietary Fats: ME, metabolism
Dinoprostone: ME, metabolism
***Fatty Acids, Unsaturated: ME, metabolism**
Inflammation: ME, metabolism
*Kupffer Cells: ME, metabolism
***Membrane Lipids: ME, metabolism**
*Phospholipids: ME, metabolism
Rats
Rats, Inbred Strains
Safflower Oil: ME, metabolism

RN 363-24-6 (Dinoprostone); 7440-70-2 (Calcium); 8001-23-8 (Safflower Oil); 8001-30-7 (Corn Oil)

CN 0 (Calcium Channels); 0 (Dietary Fats); 0 (**Fatty Acids**, Unsaturated); 0 (Membrane **Lipids**); 0 (Phospholipids)

L109 ANSWER 30 OF 30 MEDLINE on STN

AN 88291006 MEDLINE

DN PubMed ID: 3041642

TI **Fatty acid** intake and Kupffer cell function: **fish oil** alters eicosanoid and monokine production to endotoxin stimulation.

AU Billiar T R; Bankey P E; Svingen B A; Curran R D; West M A; Holman R T; Simmons R L; Cerra F B

CS Department of Surgery, University of Minnesota, Minneapolis.

SO Surgery, **(1988 Aug)** 104 (2) 343-9.
Journal code: 0417347. ISSN: 0039-6060.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 198809

ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880902

AB Diets high in **n-3 fatty acids** appear to have an anti-inflammatory effect, which is thought to be due to decreased macrophage prostaglandin (PG) and thromboxane (Tx) production after incorporation of these **fatty acids** into cell membrane phospholipids. The effect of **n-3**

fatty acids incorporation on macrophage monokine release in response to **septic** stimuli is not well established. Kupffer cells, the fixed macrophages of the liver, were obtained from rats fed diets with fat sources derived from corn oil (CO, control), **fish oil** (FO, high in **n-3 fatty acids**), or safflower oil (SO, high in **n-6 fatty acids**) for 2 or 6 weeks. After exposure to bacterial lipopolysaccharide, Kupffer cells from rats fed FO for 2 or 6 weeks produced less PG and Tx than Kupffer cells from rats fed CO or SO. After 2 weeks of defined diets, interleukin-1 (IL-1) and tumor necrosis factor release were not affected by dietary fat source. In contrast, after 6 weeks of feeding, Kupffer cells from both the FO and the SO groups released less IL-1 and tumor necrosis factor when triggered by lipopolysaccharide than Kupffer's cells from animals fed the control diet that contained CO. These data suggest that altered monokine release from macrophages may contribute to the anti-inflammatory effect of diets high in **n-3 fatty acids.** Also shown in our results is that prolonged changes in membrane phospholipid content induced by dietary fat source can influence not only PG and Tx production but monokine release as well.

CT Check Tags: Comparative Study
Animals
*Dietary Fats: PD, pharmacology
Escherichia coli
Fatty Acids, Unsaturated: AD, administration & dosage
***Fatty Acids, Unsaturated: PD, pharmacology**
Fish Oils: PD, pharmacology
Interleukin-1: BI, biosynthesis
Kupffer Cells: IM, immunology
*Kupffer Cells: PH, physiology
Lipopolysaccharides: PD, pharmacology
Lymphokines: IM, immunology
Membrane Lipids: PH, physiology
Prostaglandins: BI, biosynthesis
Rats
Rats, Inbred Strains
Thromboxanes: BI, biosynthesis
Tumor Necrosis Factor: BI, biosynthesis

CN 0 (Dietary Fats); 0 (**Fatty Acids**, Unsaturated); 0 (**Fish Oils**); 0 (Interleukin-1); 0 (Lipopolysaccharides); 0 (Lymphokines); 0 (Membrane **Lipids**); 0 (Prostaglandins); 0 (Thromboxanes); 0 (Tumor Necrosis Factor)

=> => fil wpix
FILE 'WPIX' ENTERED AT 11:22:07 ON 31 AUG 2004
COPYRIGHT (C) 2004 THOMSON DERWENT

FILE LAST UPDATED: 26 AUG 2004 <20040826/UP>
MOST RECENT DERWENT UPDATE: 200455 <200455/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE, PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf <<<

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE http://thomsonderwent.com/coverage/latestupdates/ <<<

>>> FOR INFORMATION ON ALL DERWENT WORLD PATENTS INDEX USER GUIDES, PLEASE VISIT:
http://thomsonderwent.com/support/userguides/ <<<

>>> NEW! FAST-ALERTING ACCESS TO NEWLY-PUBLISHED PATENT DOCUMENTATION NOW AVAILABLE IN DERWENT WORLD PATENTS INDEX FIRST VIEW - FILE WPIFV.
FOR FURTHER DETAILS: http://www.thomsonderwent.com/dwpifv <<<

>>> NEW DISPLAY FORMAT HITSTR ADDED ALLOWING DISPLAY OF HIT STRUCTURES WITHIN THE BIBLIOGRAPHIC DOCUMENT <<<

=> d all abeq tech abex tot

L142 ANSWER 1 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2003-416447 [39] WPIX
CR 1999-527413 [44]
DNC C2003-110187
TI **Oil**-in-water emulsion useful for treating e.g. cancer comprises **gamma linolenic** acid, **eicosapentaenoic** acid as delta-desaturase inhibitor, emulsifying agent or emulsion stabilizer and water.
DC A96 B05 D13
IN CHILTON, F H; KOUMENIS, I L; SURETTE, M E; TRAMPOSCH, K
PA (CHIL-I) CHILTON F H; (KOUM-I) KOUMENIS I L; (SURE-I) SURETTE M E; (PILO-N) PILOT THERAPEUTICS INC
CYC 102
PI US 2002188024 A1 20021212 (200339)* 50 A61K031-202
WO 2003063793 A2 20030807 (200361) EN A61K000-00
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
AU 2003208920 A1 20030902 (200422) A61K031-202
ADT US 2002188024 A1 **CIP of US 1998-28256 19980223, CIP of WO 1999-US3120 19990212**, CIP of US 2000-644380 20000823, US 2002-66334 20020131; WO 2003063793 A2 WO 2003-US2954 20030131; AU 2003208920 A1 AU 2003-208920 20030131
FDT US 2002188024 A1 CIP of US 6107334; AU 2003208920 A1 Based on WO 2003063793
PRAI US 2002-66334 20020131; **US 1998-28256 19980223; WO 1999-US3120 19990212**; US 2000-644380 20000823
IC ICM A61K000-00; A61K031-202
AB US2002188024 A UPAB: 20040331
NOVELTY - **Oil**-in-water emulsion comprises **gamma** - **linolenic** acid (GLA) (0.5 - 3, pediatric 0.2 - 3 g), **eicosapentaenoic** acid (EPA) as a Delta 5-desaturase inhibitor (0.1 - 3, pediatric 0.02 - 3 g), an emulsifying agent or emulsion stabilizer and water, where the bioavailability of the emulsified GLA and EPA is greater than that of GLA and EPA administered in gel capsule form.
ACTIVITY - Antiasthmatic; Antiallergic; **Antiinflammatory**; Antipsoriatic; Cardiant; Nephrotropic; Gastrointestinal-Gen.; Antiarthritic; Cytostatic; Dermatological; Immunosuppressive; Neuroleptic; Antidepressant; Antibacterial; Vasotropic; Nootropic; Neuroprotective; Antiarteriosclerotic; Gynecological; Analgesic; Tocolytic; Antigout; Antiulcer; Antithyroid; Muscular-Gen.; Osteopathic; Antirheumatic; Uropathic; Ophthalmological; Tranquilizer.
MECHANISM OF ACTION - Delta 5-desaturase Inhibitor; Serum **Arachidonic** Acid Increase Inhibitor.
In tests to evaluate the influence of the combination of **gamma** -**linolenic** acid (GLA) and **eicosapentaenoic** acid (EPA) on the **fatty acid** composition of serum and neutrophil **lipids**, three subjects on control diet (25% fat) were

supplemented with a combination of EPA (1.5 g/day) and GLA (3.0 g/day) for three weeks. GLA when administered alone showed accumulation of **arachidonic** acid (AA) and **dihomo- gamma - linolenic** acid (DGLA) in serum and polymorphonuclear (PMN) cells. The AA/DGLA accumulation (micro mol/5 million PMN) was found to be 0.9/0.125 (at 0 weeks), 1/0.25 (after 4 weeks), 1.125/0.25 (after 8 weeks), and 1/0.24 (after 12 weeks), respectively. The results showed that GLA alone induced marked increase in serum AA in both short (3 weeks) and the long term (12 weeks). However, the combination of GLA and EPA did not cause an increase in the serum AA levels. The **fatty acids** concentration in the serum of AA/GLA/DGLA/EPA (micro mol/l serum) was 350/20/100/20 (at 0 weeks), 350/25/105/100 (after 1 week), 450/50/250/260 (after 2 weeks) 400/40/160/100 (after 3 weeks), and 450/50/100/40 (after washout). The results showed that blocking of Delta 5-desaturase in humans provides a means to supplement humans with high levels of GLA without concomitant increase in serum AA levels.

USE - The emulsion is useful for treating a **lipid**-mediated disorder having an **arachidonic** acid metabolite component e.g. asthma, allergic rhinitis, allergic rhinoconjuctivitis, psoriasis, acute myocardial infarction, glomerulonephritis, Crohn's disease, **inflammatory** bowel disease, arthritis, breast cancer, colon cancer, prostate cancer, squamous cell carcinoma, intestinal cancer, ovarian cancer, uterine cancer, testicular cancer, autoimmune disease, systemic lupus erythematosus, schizophrenia, depression, immunoglobulin A nephropathy, **sepsis**, toxic **shock**, organ failure, organ transplant, coronary angioplasty, risk reduction for Alzheimer's disease, cystic fibrosis, atherosclerosis, atopic dermatitis, menstrual discomfort, cyclic breast pain, premature labor, early parturition, gout, venous leg ulcers, chronic urticaria, thyroiditis, primary dysmenorrhea, endometriosis, Lyme disease, muscle wasting, ankylosing spondylitis, carpal tunnel syndrome, childhood or juvenile arthritis, chronic back injury, fibromyalgia, infectious arthritis, osteoarthritis, osteoporosis, Paget's disease, polymyalgia rheumatica, polymyositis, dermatomyositis, pseudogout, psoriatic arthritis, Raynaud's syndrome, reactive arthritis, Reiter's syndrome, repetitive stress injury, rheumatoid arthritis, scleroderma and Sjogrens syndrome (all claimed). Also useful for controlling or reducing the symptoms of **inflammation**, and as a dietary and pediatric formulations.

ADVANTAGE - The bioavailability of emulsified GLA and EPA is greater than the bioavailability of GLA and EPA administered in gel form. The combination of GLA and Delta 5-desaturase inhibitor EPA prevents the increase in serum **arachidonic** acid level upon GLA administration. Additionally **omega -3**-AA is provided and the build up of **omega -3**-AA in neutrophils results in further inhibition of the serum Delta 5-desaturation of DGLA in hepatocytes, resulting in further inhibition of serum AA accumulation.

Dwg.0/25

FS CPI

FA AB; DCN

MC CPI: A12-V01; B03-F; **B04-B01B**; B04-B01C1; B04-C02A2; B04-C02B; B04-C02D; B04-N02; B05-B01P; B07-A02A; B10-C04E; B12-M03; B12-M06; B14-A01; B14-C01; B14-C02; B14-C03; B14-C09; B14-D03; B14-E08; B14-E10C; B14-E11; B14-F01B; B14-F02; B14-F04; B14-F07; B14-G02A; B14-G02C; B14-G02D; B14-H01; B14-J01A1; B14-J01A4; B14-J01B3; B14-J01B4; B14-J05; B14-K01; B14-N01; B14-N03; B14-N04; B14-N07; B14-N10; B14-N11; B14-N12; B14-N14; B14-N16; B14-N17; B14-N18; B14-P03; **B14-S06**; B14-S09; D03-A; D03-H01A; D03-H01C; D03-H01E; D03-H01N; **D03-H01T2**; D03-H02

TECH UPTX: 20030619

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Formulation: The formulation additionally comprises at least one flavoring agent, sweetening agent, coloring agent or a preservative.

Preferred Method: The administration of the emulsion alters the synthesis

of at least one **arachidonic** acid (AA) metabolite.
Preferred Components: The AA metabolite is at least one of leukotrienes (LT), prostaglandins (PG) or lipoxins (LX) (preferably 5-hydroperoxy-eicosatetraenoic acid, 5-hydroxyeicosatetraenoic acid (5-HETE), 5-HETE-lactone, leukotriene A4 (LTA4), 5(S),6(S)-dihydroxyeicosatetraenoic acid (DIHETE), 5(S),6(R)-DIHETE, LTB4, lipoxin A4 (LXA4), LTC4, 12(R,S),6-trans-LTB4, LTF4, LTD4, LTE4, 20-OH-LTE4, 20-COOH-LTE4, 18-COOH-LTE4, 16-COOH-LTE3, 14-COOH-LTE3, LTE4-Nac, prostaglandin G2 (PGG2), PGH2, PGD2, 13,14-dihydra-15-keto-PGD2, 9-alpha,11-beta-PGF2, PGJ2, DELTA12-PGJ2, PGE2, 9alpha,11alpha-PGF2, PGA2, PGB2, 19-OH-PGE2, 15-keto-PGE2, 13,14-dihydro-15-keto-PGE2, PGE-M, PGF2alpha, 15-keto-PGF2alpha, 13,14-dihydro-15-keto-PGF2alpha, PGF-M, lipoxin A4, lipoxin B4, 15-epi-lipoxin A4 or 15-epi-lipoxin A5; especially leukotriene; particularly LTB4).

TECHNOLOGY FOCUS - POLYMERS - Preferred Components: The emulsifying agent or emulsion stabilizer is xanthan gum, guar gum, pectin, carob seed gum (locust-bean gum), tragacanth gum, methylcellulose, starch, modified starch, carboxymethylcellulose, gum Arabic or gelatin, **phospholipid**, lecithin, alginates or carrageenan.

ABEX UPTX: 20030619
ADMINISTRATION - The emulsion is administered in a unit dosage form, providing a daily dosage of EPA and GLA (claimed). The emulsion is administered orally. The dosage comprises GLA 1 - 15 (preferably 1.5 - 3) g; EPA 0.1 - 10 (preferably 0.5 - 3) g, and optionally stearidonic acid 0.1 - 15 (preferably 3 - 5) g, administered 1 - 4 times daily. The dosage of GLA is 0.2 - 3, and of EPA is 0.02 - 3, when used as a pediatric formulation.
EXAMPLE - A dietary fatty acid emulsion was prepared as follows. A stabilized emulsion comprised (g): borage **oil** (21), marine **oil** (16.5), lecithin (0.5), flavor and flavor masking agent (2), colorant (0.05), ascorbyl palmitate (0.2), sorbic acid (0.16), sucrose (25), xanthan gum (0.3), water (29.29) and glycerin (5). The emulsion prepared was preferably packaged in an oxygen-free environment in single daily dosage packages made of oxygen impermeable materials, such as foil-lined pouches. A dosage of 20 g/day was recommended, along with **gamma-linolenic** acid (1.5 g) and **eicosapentaenoic** acid (1.0 g).

L142 ANSWER 2 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 2001-281492 [29] WPIX
DNC C2001-085515
TI Composition for use as a medicament, food or nutritive product, for the treatment of **sepsis** or **inflammatory shock**, comprises at least one **lipid.**
DC B05 C03 D13
IN BREUILLE, D; CROZIER-WILLI, G; DUTOT, G; FINOT, P; OBLED, C; RICHELLE, M; ROESSLE, C; TURINI, M
PA (INRG) INRA INST NAT RECH AGRONOMIQUE; (NEST) SOC PROD NESTLE SA
CYC 95
PI WO 2001019356 A2 20010322 (200129)* EN 29 A61K031-00
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
EP 1090636 A1 20010411 (200129) EN A61K031-23
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
AU 2000068430 A 20010417 (200140) A61K031-00
BR 2000013958 A 20020514 (200240) A61K031-00
EP 1216041 A2 20020626 (200249) EN A61K031-23

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

CN 1390125 A 20030108 (200334) A61K031-23
MX 2002002728 A1 20020901 (200370) A61K031-00
EP 1216041 B1 20040204 (200410) EN A61K031-23
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
DE 60008127 E 20040311 (200419) A61K031-23
ES 2213036 T3 20040816 (200455) A61K031-23

ADT WO 2001019356 A2 **WO 2000-EP8731 20000907**; EP 1090636 A1 **EP 1999-118173 19990913**; AU 2000068430 A AU 2000-68430 20000907; BR 2000013958 A BR 2000-13958 20000907, **WO 2000-EP8731 20000907**; EP 1216041 A2 EP 2000-956522 20000907, **WO 2000-EP8731 20000907**; CN 1390125 A CN 2000-815576 20000907; MX 2002002728 A1 **WO 2000-EP8731 20000907**, MX 2002-2728 20020313; EP 1216041 B1 EP 2000-956522 20000907, **WO 2000-EP8731 20000907**; DE 60008127 E DE 2000-00008127 20000907, EP 2000-956522 20000907, **WO 2000-EP8731 20000907**; ES 2213036 T3 EP 2000-956522 20000907

FDT AU 2000068430 A Based on WO 2001019356; BR 2000013958 A Based on WO 2001019356; EP 1216041 A2 Based on WO 2001019356; MX 2002002728 A1 Based on WO 2001019356; EP 1216041 B1 Based on WO 2001019356; DE 60008127 E Based on EP 1216041, Based on WO 2001019356; ES 2213036 T3 Based on EP 1216041

PRAI **EP 1999-118173 19990913**

IC ICM A61K031-00; A61K031-23
ICS **A23L001-30**; A61P003-00

AB WO 200119356 A UPAB: 20010528

NOVELTY - Composition for use as a medicament, food or nutritive product, comprises at least one **lipid**, which provides more than 35 % total energy of the composition.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for the preparation of a composition as above comprising blending the constituents, liquefying the blended mixture and homogenizing.

ACTIVITY - **Antiinflammatory**; antibacterial; immunosuppressive.

Tests were performed on an animal model of **sepsis** in rats to evaluate the body weight gain of rats post infection. Rats were fed either a 15 % (INF-15) **lipid** diet or a 35 % (INF-35) **lipid** diet for 6 days prior to infection and 10 days post infection. Two uninfected test groups were also fed a 15 % (PF-15) or 35 % (PF-35) **lipid** diets. Results are shown in the figure.

MECHANISM OF ACTION - None given.

USE - The composition is used in medicaments, foods or nutritive products for the treatment and prevention of **sepsis** or **inflammatory shock.** It may also be fed to animals e.g. pets.

ADVANTAGE - The composition can provide a high **lipid** diet to patients suffering from **inflammatory shock** or **sepsis.** The composition has a beneficial effect on recovery from acute **inflammatory** stress, and also on clinical parameters (body weight loss and nitrogen excretion).

DESCRIPTION OF DRAWING(S) - The figure shows the results of tests performed on an animal model of **sepsis** in rats to evaluate the body weight gain of rats fed a 15 or 35 % **lipid** diet, post infection.

Dwg.3/13

FS CPI
FA AB; GI; DCN
MC CPI: **B04-B01B**; B10-C04E; B14-C03; **B14-S06**; **C04-B01B**; C10-C04E; C14-C03; **C14-S06**; D03-G01; **D03-H01T2**

TECH UPTX: 20010528

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Composition: The **lipid** comprises 25-70 wt. % medium chain **triglycerides**

(MCT). The **lipid** comprises less than 15 wt. % saturated **fatty acids** excluding MCT. The composition comprises a **n-6/n-3 fatty acid** ratio of 2/1 to 7/1. The composition comprises at least one **n-3 fatty acid** selected from **alpha-linolenic** acid, **eicosapentaenoic** acid (EDA), **docosapentaenoic** acid (DPA) and **docosahexaenoic** acid (DHA). The composition comprises at least one **n-6 fatty acid** selected from **linoleic** acid (18:2), **gamma-linolenic** acid (18:3), **dihomo-gamma-linolenic** acid (18:4), or **arachidonic** acid. The composition may be administered enterally and further comprise a carrier, diluent or adjuvant.

ABEX UPTX: 20010528

ADMINISTRATION - Administration is enteral (claimed).

EXAMPLE - A typical composition was prepared comprising 18 % protein, 37 % carbohydrate, and 45 % **lipid.** The **lipid** part of the composition contained (g/L): saturated **lipid** (including medium chain **triglycerides**) (42.8); monounsaturated **lipid** (23.3); polyunsaturated **lipid** (9.0); **linoleic** acid (18:2; n-6) (6.8); and **alpha-linolenic** acid (18:3; n-3) (2.3). The caloric density of the composition was 1.5 Kcal/ml.

L142 ANSWER 3 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1999-551195 [46] WPIX
DNC C1999-160839
TI Use of **omega 3 fatty acid** for treating disorders in which **docosahexaenoic** acid levels are affected.
DC B05
IN ALVAREZ, J G; FREEDMAN, S
PA (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT
CYC 82

PI	WO 9945916	A2	19990916 (199946)*	EN	50	A61K031-20	<--
	RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW						
	W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW						
	AU 9929824	A	19990927 (200006)			A61K031-20	<--
	BR 9908697	A	20001121 (200065)			A61K031-20	
	EP 1061911	A2	20001227 (200102)	EN		A61K031-20	
	R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI						
	US 6180671	B1	20010130 (200108)			A61K031-20	
	JP 2002506026	W	20020226 (200219)		67	A61K031-202	
	MX 2000008475	A1	20011101 (200279)			A23L001-30	<--
	US 6552081	B1	20030422 (200330)			A61K031-20	
	AU 762660	B	20030703 (200354)			A61K031-20	
	US 2004127567	A1	20040701 (200444)			A61K031-202	

ADT WO 9945916 A2 **WO 1999-US4722 19990303**; AU 9929824 A **AU 1999-29824 19990303**; BR 9908697 A **BR 1999-8697 19990303, WO 1999-US4722 19990303**; EP 1061911 A2 **EP 1999-911098 19990303, WO 1999-US4722 19990303**; US 6180671 B1 **CIP of US 1998-37222 19980310, CIP of US 1999-231479 19990114, US 1999-248471 19990211**; JP 2002506026 W **WO 1999-US4722 19990303, JP 2000-535331 19990303**; MX 2000008475 A1 MX 2000-8475 20000830; US 6552081 B1 **CIP of US 1998-37222 19980310, CIP of US 1999-231479 19990114, Div ex US 1999-248471 19990211**, US 2000-706404 20001103; AU 762660 B **AU 1999-29824 19990303**; US 2004127567 A1 **CIP of US 1998-37222 19980310,**

CIP of US 1999-231479 19990114, Div ex US 1999-248471 19990211, Div ex US 2000-706404 20001103, US 2003-410511 20030408

FDT AU 9929824 A Based on WO 9945916; BR 9908697 A Based on WO 9945916; EP 1061911 A2 Based on WO 9945916; JP 2002506026 W Based on WO 9945916; US 6552081 B1 Div ex US 6180671; AU 762660 B Previous Publ. AU 9929824, Based on WO 9945916; US 2004127567 A1 Div ex US 6180671, Div ex US 6552081

PRAI **US 1999-248471 19990211; US 1998-37222 19980310; US 1999-231479 19990114**; US 2000-706404 20001103; US 2003-410511 20030408

IC ICM **A23L001-30**; A61K031-20; A61K031-202
ICS A61K031-23; A61P001-00; A61P001-04; A61P011-06; A61P029-00; A61P043-00

AB WO 9945916 A UPAB: 19991110
NOVELTY - Treatment of disorders in which **docosahexaenoic** acid (DHA) levels are affected comprises administering an **omega 3 fatty acid** having 22-24 C atoms and 5 or more double bonds.

USE - The **fatty acid** is administered to treat disorders in which DHA levels are affected e.g. defects in the CF or CFTR gene, cystic fibrosis, a chronic **inflammatory** disorder e.g. ulcerative colitis, Crohn's disease, chronic pancreatitis, asthma, rheumatoid arthritis, chronic gastritis, lowered fetal surfactant levels, respiratory distress syndrome, hypertrophy of the small intestine or to restore normal morphology to a cell or tissue which exhibits a disease morphology associated with disorder in which DHA levels are affected. The method is especially useful for ameliorating the effects of cystic fibrosis in a newborn.

Dwg.5/8

FS CPI
FA AB; GI; DCN
MC CPI: B10-C04E; B14-C03; B14-C06; B14-C09; B14-E08; B14-E12; B14-G02A; B14-G02D; B14-K01; B14-K01D
TECH UPTX: 19991110
TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Compounds: The **omega 3 fatty acid** is DHA or a DHA precursor e.g. **docosapentaenoic** acid, tetracosapentaenoic acid and tetracosahexaenoic acid. The acid may be in a form easily incorporated into the tissue such as a derivative or a structured **lipid.** Preferably the structured **lipid** has the DHA located at the R2 position of the glycerol backbone.
ABEX UPTX: 19991110
ADMINISTRATION - Administration is preferably oral. DHA levels are raised to at least about 170 (preferably 200-500) g/ml. The DHA is administered at about 0.3-5% of the total calorific intake and should produce a pancreatic **arachidonic** acid (AA)/DHA ratio of about 0.2-1.5 or a lung AA/DHA ratio of 0.1-1.6 or a blood AA/DHA ratio of 0.1-1.6.

EXAMPLE - The figures show the effects of oral DHA administration at various concentrations on **arachidonic** acid (AA) and DHA levels in preparations of pancreatic acini and lung cells form CFTR (-/-) mice (CF) compared with wild type mice (WT).

L142 ANSWER 4 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1999-372252 [32] WPIX
DNC C1999-110070
TI Fat mixture with polyunsaturated fats and **oils**, useful for treating e.g. **inflammatory** diseases.
DC B05 D13
IN BOEHN, G; FARWER, S; KLIEM, M; KOHN, G; SAWATZKI, G; BOEHM, G
PA (NUTR-N) NUTRICIA NV
CYC 34
PI DE 19757414 A1 19990701 (199932)* 10 A23L001-29 <--
WO 9933355 A2 19990708 (199934) GE A23L001-00 <--

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AL AU BR CA CN IN JP LT LV MK NO NZ RO SI US

AU 9924162	A	19990719	(199951)		A23L001-00	<--
NO 2000003265	A	20000622	(200050)		A23D000-00	
EP 1041896	A2	20001011	(200052)	GE	A23L001-30	<--
R: AT BE CH DE DK ES FR GB IE IT LI NL SE						
BR 9814467	A	20001010	(200055)		A23L001-00	<--
CN 1282223	A	20010131	(200131)		A23L001-30	<--
JP 2001526908	W	20011225	(200204)	23	A23D009-007	
EP 1041896	B1	20020220	(200214)	GE	A23L001-30	<--
R: AT BE CH DE DK ES FR GB IE IT LI NL SE						
DE 59803153	G	20020328	(200229)		A23L001-30	<--
ES 2173673	T3	20021016	(200279)		A23L001-30	<--
NZ 505608	A	20021220	(200309)		A23L001-00	<--
AU 758079	B	20030313	(200328)		A23L001-00	<--
NO 316302	B1	20040112	(200406)		A23D009-00	

ADT DE 19757414 A1 **DE 1997-1057414 19971223**; WO 9933355 A2 **WO 1998-EP8409 19981222**; AU 9924162 A **AU 1999-24162 19981222**; NO 2000003265 A **WO 1998-EP8409 19981222**, NO 2000-3265 20000622; EP 1041896 A2 **EP 1998-966662 19981222, WO 1998-EP8409 19981222**; BR 9814467 A **BR 1998-14467 19981222, WO 1998-EP8409 19981222**; CN 1282223 A **CN 1998-812474 19981222**; JP 2001526908 W **WO 1998-EP8409 19981222, JP 2000-526127 19981222**; EP 1041896 B1 **EP 1998-966662 19981222, WO 1998-EP8409 19981222**; DE 59803153 G **DE 1998-503153 19981222 , EP 1998-966662 19981222, WO 1998-EP8409 19981222**; ES 2173673 T3 **EP 1998-966662 19981222**; NZ 505608 A **NZ 1998-505608 19981222, WO 1998-EP8409 19981222**; AU 758079 B **AU 1999-24162 19981222**; NO 316302 B1 **WO 1998-EP8409 19981222**, NO 2000-3265 20000622

FDT AU 9924162 A Based on WO 9933355; EP 1041896 A2 Based on WO 9933355; BR 9814467 A Based on WO 9933355; JP 2001526908 W Based on WO 9933355; EP 1041896 B1 Based on WO 9933355; DE 59803153 G Based on EP 1041896, Based on WO 9933355; ES 2173673 T3 Based on EP 1041896; NZ 505608 A Based on WO 9933355; AU 758079 B Previous Publ. AU 9924162, Based on WO 9933355; NO 316302 B1 Previous Publ. NO 2000003265

PRAI **DE 1997-19757414 19971223**

IC ICM A23D000-00; A23D009-00; A23D009-007; **A23L001-00**; **A23L001-29**; **A23L001-30**
ICS A61K031-20; A61K045-00; A61P003-06; A61P029-00; A61P037-04

AB DE 19757414 A UPAB: 19990813
NOVELTY - A fat mixture (I) comprising **oils**, fats and/or lecithins with polyunsaturated fatty acids which comprise **gamma -linolenic** acid (20-50 weight%), stearidonic acid (15-50 weight%) and **eicosapentaenoic** acid (20-50 weight%) at 10-500 mg/g.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for dietary foods containing (I).
USE - (I) is used in the dietary treatment of patients with chronic **inflammatory** diseases, disturbances of **lipid** metabolism, weak immune system function and/or limited lipolytical capacity of the gastrointestinal tract (claimed).
Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: **B04-B01B**; B10-C04E; B14-C03; B14-E10; B14-G01; D03-C01; **D03-H01T2**

TECH UPTX: 19990813
TECHNOLOGY FOCUS - FOOD - Preferred Materials: (I) comprises **gamma -linolenic** acid and **eicosapentaenoic** acid (35-45 wt.%) and stearidonic acid (15-25 wt.% of the total of three acids). (I) comprises **phospholipids** (1-10 wt.% of the total **lipids**). The dietary foods are in the form of fatty emulsions, ready made meals, liquid food, reconstituted or reconstitutable powder, especially for

enteral or oral application, as bar or spread. The dietary food comprises an energy supply of 0.5-3 kcal/ml.

L142 ANSWER 5 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1998-178507 [16] WPIX
DNC C1998-057272
TI Enteral nutrient compositions for optimised absorption and wound healing in e.g. trauma patients - comprising partially hydrolysed proteins, free amino acids, carbohydrates and **lipids** including **omega-3 fatty acids.**
DC B05 D13
IN ALEXANDER, J; GRAY, D; MARK, D A; SCHMELKIN, N S; TWYMAN, D
PA (NEST) NESTEC LTD
CYC 1
PI US 5723446 A 19980303 (199816)* 5 A61K038-00 <--
ADT US 5723446 A **Cont of US 1993-172857 19931223, US 1996-680703 19960717**
PRAI **US 1993-172857 19931223; US 1996-680703 19960717**
IC ICM A61K038-00
ICS A23G003-00; A23J001-00
AB US 5723446 A UPAB: 19980421
Providing enteral nutrition to trauma, burn or post-surgery patients comprises enterally administering a composition containing: (a) a protein source comprising 22-28(25)% of the total calories; (b) a carbohydrate source comprising 35-40% of the total calories; and (c) a **lipid** source comprising 33-45% of the total calories including a source of medium chain **triglycerides** comprising 40-60% of the **lipid** source, a source of **omega -3 fatty acids** providing 2.2-3% of the total calories, and a source of **omega -6 fatty acids.**
The composition provides sources of arginine, proline, beta -carotene and/or cysteine. The protein source includes a majority(68-88%) of protein calories as partially hydrolysed proteins and does not contain whole proteins. The composition is fed to a patient through a tube and has a caloric density of 1.3-1.5 kilocalories/ml.
ADVANTAGE - The composition has high protein and **lipid** content and high caloric density to meet energy needs, and has reduced water and carbohydrate content, reducing the risk of diarrhea. Nutrient malabsorption is reduced by the absence of whole proteins and by the use of protein hydrosylate, free amino acids and medium chain **triglycerides.** The ready to use formulation does not require mixing, thus reducing the risk of bacterial contamination. The composition minimises **inflammatory** reactions.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B03-A; **B04-B01B**; B04-D01; B04-N02; B05-B01P; B07-D03; B10-A17; B10-B02D; B10-C04E; B10-G02; B14-E11; B14-N17; D03-F06; **D03-H01T2**

L142 ANSWER 6 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1997-299573 [28] WPIX
DNC C1997-097229
TI Isotonic **lipid** emulsion for parenteral application for nourishment, - in patients with, e.g. exacerbated **inflammatory** reaction or increased risk of vascular thrombosis, based on plant and **fish oil** and middle-chain tri **glyceride** derivatives.
DC B04 D13
IN CARPENTIER, Y A; JUNGINGER, M; NEHNE, J; PSCHERER, G; JUNINGER, M
PA (BINT) BRAUN MELSUNGEN AG B
CYC 66

PI DE 19648566 A1 19970605 (199728)* 13 A61K035-78 <--
WO 9719683 A1 19970605 (199728) EN 32 A61K031-19 <--
RW: AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG
W: AL AU BA BB BG BR CA CN CU CZ EE GE HU IL IS JP KP KR LC LK LR LT LV MG MK MN MX NO NZ PL RO SG SI SK TR TT UA US UZ VN
AU 9676972 A 19970619 (199741) A61K031-19 <--
EP 863754 A1 19980916 (199841) EN A61K031-19 <--
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE SI
CZ 9801618 A3 19981111 (199851) A61K031-19 <--
AU 701736 B 19990204 (199917) A61K031-19 <--
CN 1202823 A 19981223 (199919) A61K031-19 <--
NZ 322927 A 19990629 (199931) A23L001-30 <--
BR 9611826 A 19990713 (199939) A61K031-19 <--
US 6008248 A 19991228 (200007) A61K031-19 <--
JP 2000500769 W 20000125 (200016) 28 A61K031-201
HU 9903426 A2 20000228 (200020) A61K031-19
MX 9804127 A1 19981201 (200024) A61K031-19 <--
CA 2236422 C 20021119 (200304) EN A61K031-19
EP 863754 B1 20030102 (200310) EN A61K031-19
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE SI
DE 69625616 E 20030206 (200318) A61K031-19
ES 2189891 T3 20030716 (200356) A61K031-19
MX 213257 B 20030317 (200413) A23L001-30 <--

ADT DE 19648566 A1 **DE 1996-1048566 19961123**; WO 9719683 A1 **WO 1996-EP5184 19961123**; AU 9676972 A **AU 1996-76972 19961123**; EP 863754 A1 **EP 1996-939912 19961123, WO 1996-EP5184 19961123**; CZ 9801618 A3 **WO 1996-EP5184 19961123, CZ 1998-1618 19961123**; AU 701736 B **AU 1996-76972 19961123**; CN 1202823 A **CN 1996-198603 19961123**; NZ 322927 A **NZ 1996-322927 19961123, WO 1996-EP5184 19961123**; BR 9611826 A **BR 1996-11826 19961123, WO 1996-EP5184 19961123**; US 6008248 A **WO 1996-EP5184 19961123, US 1998-43166 19980312**; JP 2000500769 W **WO 1996-EP5184 19961123, JP 1997-520149 19961123**; HU 9903426 A2 **WO 1996-EP5184 19961123 , HU 1999-3426 19961123**; MX 9804127 A1 **MX 1998-4127 19980525**; CA 2236422 C **CA 1996-2236422 19961123, WO 1996-EP5184 19961123**; EP 863754 B1 **EP 1996-939912 19961123, WO 1996-EP5184 19961123**; DE 69625616 E **DE 1996-625616 19961123, EP 1996-939912 19961123, WO 1996-EP5184 19961123**; ES 2189891 T3 **EP 1996-939912 19961123**; MX 213257 B **WO 1996-EP5184 19961123, MX 1998-4127 19980525**

FDT AU 9676972 A Based on WO 9719683; EP 863754 A1 Based on WO 9719683; CZ 9801618 A3 Based on WO 9719683; AU 701736 B Previous Publ. AU 9676972, Based on WO 9719683; NZ 322927 A Based on WO 9719683; BR 9611826 A Based on WO 9719683; US 6008248 A Based on WO 9719683; JP 2000500769 W Based on WO 9719683; HU 9903426 A2 Based on WO 9719683; CA 2236422 C Based on WO 9719683; EP 863754 B1 Based on WO 9719683; DE 69625616 E Based on EP 863754, Based on WO 9719683; ES 2189891 T3 Based on EP 863754

PRAI **DE 1995-19544310 19951128**

REP DE 3409793; DE 3721137; EP 311091; EP 687418; FR 2542613; US 5444054; US 5470839

IC ICM A61K031-19; A61K031-201; A61K035-78
ICS A61K009-107; A61K031-23; A61K031-231; A61K035-60; A61K045-00; A61P003-02; A61P005-30; A61P007-02; A61P009-00; A61P029-00

ICA A23D007-00; **A23L001-30**

AB DE 19648566 A UPAB: 19970709
Isotonic **lipid** emulsion for parenteral application consisting of middle chain **triglycerides** (30-60 weight%) and at least 1 plant **oil** containing **omega-6-fatty acids** (35-65 weight%) and a **fish oil** containing **omega-3-fatty acids** (5-20 weight%).
USE - The composition is used for parenteral nourishment in patients

with exacerbated **inflammatory** reaction, increased risk of vascular thrombosis, heart arrhythmia, post operative or post traumatic conditions or **inflammatory** disease (all claimed).
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B04-B01C1; B04-B01C2; B12-M03; B14-C03; B14-F01A; B14-F04; **D03-H01T2**

L142 ANSWER 7 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1997-021672 [02] WPIX
CR 1996-506074 [50]; 1996-506079 [50]; 1996-506082 [50]; 1997-021670 [49]; 1997-021671 [49]
DNC C1997-007027
TI gem-Diol ester(s) of polyunsaturated fatty acids e.g. **gamma linolenic** acid - for pharmaceutical, food and cosmetic use.
DC B04 B05 C03 D13 D21
IN HORROBIN, D F; KNOWLES, P; MANKU, M; MCMORDIE, A; PITT, A; REDDEN, P
PA (SCOT-N) SCOTIA HOLDINGS PLC
CYC 1
PI ZA 9603433 A 19961030 (199702)* EN 41 A23L000-00 <--
ADT ZA 9603433 A **ZA 1996-3433 19960430**
PRAI **GB 1995-8823 19950501**
IC ICM A23L000-00
ICS A61K000-00; C07C000-00
AB ZA 9603433 A UPAB: 19970212
gem-Diol esters of formula R1O-CHR3-OR2 (I) are new. R1 = acyl gp. derived from a 16-30C **fatty acid** containing two or more cis or trans double bonds, partic. an **n-6** or **n-3** series essential **fatty acid** or conjugated **linoleic** acid (cLA) or columbinic acid (CA) or parinaric acid; R2 = R1 or any other nutrient, drug or bioactive residue; R3 = H or hydrocarbyl opt. containing heteroatoms, pref. alkyl, partic. 1-4C alkyl.
USE - (I) where R1 = acyl derived from **gamma linolenic** acid (GLA) or **dihomo gamma linolenic** acid (DGLA) and R2 = acyl derived from GLA, DGLA, stearidonic acid (SA), **eicosapentaenoic** acid (EPA), **docosahexaenoic** acid (DHA), cLA or CA are useful as food components, nutritional supplements, food additives, components of clinical nutrition prods. for enteral or parenteral admin. and cosmetic components, especially for treating (a) complications of diabetes, especially neuropathy and retinopathy; and improvement of responses to insulin in diabetes and pre-diabetes; (b) cancers; (c) osteoarthritis; (d) rheumatoid arthritis; (e) other **inflammatory** and auto-immune diseases e.g. Sjogren's syndrome, systemic lupus, ulcerative colitis, Crohn' s disease and uveitis; (f) respiratory diseases e.g. asthma; (g) neurological disorders e.g. multiple sclerosis, Parkinson's disease and Huntington' s chorea; (h) renal and urinary tract disorders; (i) cardiovascular disorders; (j) degenerative diseases of the eye e.g. retinitis pigmentosa and senile macular degeneration; (k) psychiatric disorders including schizophrenia, Alzheimer's disease, attention deficit disorder, alcoholism and depression; (l) prostatic hypertrophy and prostatitis; (m) impotence and male infertility; (n) mastalgia; (o) male pattern baldness; (p) osteoporosis; (q) dermatological and allergic disorders; (r) dyslexia and other learning disabilities; and (s) cancer cachexia. (I) where R1 = acyl derived from GLA, DGLA, **arachidonic** acid (AA), SA, cLA, EPA or DHA and R2 = one of the following agents are useful for treating any disease especially the following disorders, and other uses mentioned: (a) tryptophan for psychiatric, neurological, behavioural, pain and other disorders and especially depression, sleep and migraine; (b) phenylalanine for depression, multiple sclerosis and chronic fatigue syndrome; (c) arginine for diseases in which the production of nitric oxide is defective; (d) carnitine or carnitine derivs. for muscle weakness, cardiac failure,

chronic fatigue syndrome, Alzheimer's disease, and peripheral neuropathies; (e) any other amino acid or related substance or aminolevulinic acid or derivative thereof for cancers; (f) adenylosuccinate or related substances for muscular dystrophy , cardiac failure, chronic fatigue and Alzheimer' s disease and other dementias; (g) aspirin, salicylic acid, indomethacin, ibuprofen, or any other non-steroidal anti-**inflammatory** drug for **inflammatory** disorders or pain, of Alzheimer's disease and other dementias and of any disease in which platelet aggregation should be inhibited; (h) any antibiotic for the treatment of any appropriate infectious disease but especially tetracycline, clindamycin, minocycline, chlortetracycline and erythromycin for the treatment of acne; (i) any antimalarial or anti-protozoal drug especially chloroquine, mepacrine, quinacrine and mefloquine for the treatment of malaria, protozoal disorders, **inflammatory** disorders and schizophrenia; (j) any antifungal drug especially metronidazole and antifungal imidazoles and nitroimidazoles and amphotericin for the treatment of fungal infections of various types; (k) any anti-**inflammatory** steroid especially hydrocortisone and betamethasone for the treatment of skin disorders and beclomethasone and budesonide for the treatment of asthma; (l) any gonadal steroid especially oestrogens and progestogens for the treatment of ovarian deficiency and osteoporosis and androgens for the treatment of testicular deficiency; (m) any adrenal steroid especially dehydroepiandrosterone for the treatment of disorders associated with ageing; (n) any retinoid especially tretinoin and isotretinoin for the treatment of dermatological disorders and for use in skin care ; (o) any anticancer agent for the treatment of cancer; (p) any antipsychotic agent for the treatment of schizophrenia and other psychoses; (q) any antidepressive agent for the treatment of depression; (r) any anti-anxiety agent especially for the treatment of anxiety and panic attacks; (s) any immunosuppressive agent especially cyclosporine and tacrolimus for the control of immunity after organ transplantation and for the treatment of autoimmune and **inflammatory** disorders including psoriasis, eczema, asthma, rheumatoid arthritis and **inflammatory** bowel disease; (t) any proton pump inhibitor or H2 antagonist especially diseases associated with excess gastric acid production or reduced defences against gastric acidity; (u) any diuretic to treat fluid retention and hypertension; (v) any calcium antagonist or angiotensin converting enzyme inhibitor or beta blocker to treat cardiovascular disease; (w) antiepileptic drug especially phenytoin, carbamazepine or lamotrigine to treat epilepsy; (x) any **hypolipidaemic** agent especially fibrates and statins for cholesterol lowering; (y) any oral hypoglycaemic for diabetes management; (z) any bisphosphonates for management of osteoporosis or Paget's disease; (aa) any contrast agents for radiology; (bb) any peptide or protein for treatment using these diseases.

ADVANTAGE - Transport through **lipid** membranes, e.g. of cells, of the skin or the blood-brain barrier, is enhanced.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B10-G02; C10-G02; B14-C03; C14-C03; B14-C09A; C14-C09A; B14-C09B; C14-C09B; B14-E10C; C14-E10C; B14-E11; C14-E11; B14-G02D; C14-G02D; B14-H01; C14-H01; B14-J01A; C14-J01A; B14-K01A; C14-K01A; B14-N03; C14-N03; B14-N07A; C14-N07A; B14-S04; C14-S04; **D03-H01T2**; D08-B09A

L142 ANSWER 8 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1996-031549 [04] WPIX

DNC C1996-010844

TI **Lipid** compsn. for limiting injury response in trauma, burns etc. - comprises medium chain tri **glyceride** and **omega**-

6 and -3 **fatty acid** sources, useful for enteral and parenteral admin..

DC B05 D13
IN TRIMBO, S; TRIMBO, S L
PA (CLIN-N) CLINTEC NUTRITION CO; (NEST) SOCIETE DES PRODUITS NESTLE SA; (NEST) NESTEC LTD
CYC 18
PI EP 687418 A2 19951220 (199604)* EN 5 A23D009-00 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU NL PT SE
CA 2147302 A 19951022 (199610) A61K031-22 <--
US 5574065 A 19961112 (199651) 5 A61K031-22 <--
EP 687418 A3 19970514 (199731) A23D009-00 <--
US 5700837 A 19971223 (199806) 6 A61K031-22 <--
ADT EP 687418 A2 **EP 1995-200987 19950418**; CA 2147302 A **CA 1995-2147302 19950419**; US 5574065 A **US 1994-230592 19940421** ; EP 687418 A3 **EP 1995-200987 19950418**; US 5700837 A **Cont of US 1994-230592 19940421, US 1996-612980 19960305**
FDT US 5700837 A Cont of US 5574065
PRAI **US 1994-230592 19940421; US 1996-612980 19960305**
REP DE 3721137; EP 311091; EP 484266; US 5444054; US 5470839
IC ICM A23D009-00; A61K031-22
ICS **A23L001-30**; A61K031-00; A61K031-19; A61K031-20; A61K031-225
AB EP 687418 A UPAB: 19970522
A **lipid** compsn. comprises medium chain **triglyceride**, an **omega -6 fatty acid** source and an **omega -3 fatty acid** source.
USE - The compsns limit the injury response in patients suffering from trauma, burns and/or **sepsis.** They may be part of a complete diet and may be in the form of a parenteral emulsion or an enteral compsn.
Dwg.0/0
FS CPI
FA AB; DCN
MC CPI: B04-B01C1; B04-B01C2; B10-G02; B14-N17A; B14-N17B; **B14-S06**; **D03-H01T2**
ABEQ US 5574065 A UPAB: 19961219
Lipid compsn. comprises:
medium chain **triglycerides**, the medium chain **triglycerides** comprise approx. 25 wt.% of the compsn.;
an **omega-3 fatty acid** source,
the **omega-3 fatty acid** source comprises approx. 35 wt.% of the compsn., and
an **omega-6 fatty acid** source,
the **omega-6 fatty acid** source comprises approx. 40 wt.% of the compsn.
Dwg.0/0
ABEQ US 5700837 A UPAB: 19980209
A **lipid** compsn. comprises medium chain **triglyceride**, an **omega -6 fatty acid** source and an **omega -3 fatty acid** source.
USE - The compsns limit the injury response in patients suffering from trauma, burns and/or **sepsis.** They may be part of a complete diet and may be in the form of a parenteral emulsion or an enteral compsn.
Dwg.0/0

L142 ANSWER 9 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1995-310360 [40] WPIX
CR 1995-358387 [46]; 1999-589273 [50]; 2003-127890 [12]
DNC C1995-138367
TI Treatment of ulcerative colitis and/or colon **inflammation** - using nutritional prod. containing blend of **n-6** and **n-3 fatty acids** with carbohydrate metabolised to short chain **fatty acids** in colon.

DC B05 D13
IN DEMICHELE, S J; GARLEB, K A; FULLER, M K; MCEWEN, J W
PA (ABBO) ABBOTT LAB
CYC 20
PI US 5444054 A 19950822 (199540)* 23 A61K031-70 <--
EP 754001 A1 19970122 (199709) EN A23L001-30 <--
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
BR 1101101 A3 19980512 (199828) A61K031-20 <--
MX 9605144 A1 19971201 (199936) A23L001-30 <--
MX 192837 B 19990730 (200061) A61K031-715 <--
ES 2171535 T3 20020916 (200270) A23L001-30 <--
ADT US 5444054 A **US 1994-221440 19940401**; EP 754001 A1 **EP 1995-911135 19950306, WO 1995-US2657 19950306**; BR 1101101 A3 **BR 1997-1101101 19970514**; MX 9605144 A1 **MX 1996-5144 19961025**; MX 192837 B **MX 1996-5144 19950306**; ES 2171535 T3 **EP 1995-911135 19950306**
FDT EP 754001 A1 Based on WO 9526646; ES 2171535 T3 Based on EP 754001
PRAI **US 1994-221440 19940401; US 1994-221349 19940401; WO 1995-US2657 19950306**
REP 2.Jnl.Ref; EP 115419; EP 404058; JP 4066528; US 5104677; WO 9012080; WO 9321912
IC ICM **A23L001-30**; A61K031-20; A61K031-70; A61K031-715
ICS A23G003-000; **A23L001-305**; **A23L001-308**
AB US 5444054 A UPAB: 20030218
Method of improving the nutritional status and reversing the characteristic diarrhoea and **inflammatory** condition in a mammal having ulcerative colitis or **inflammation** of the colon is claimed. The method comprises feeding the patient enterally with a liquid nutritional prod. (A1) or (A2). (A1) comprises (a) an **oil** blend (a1) or (a'1) and a source of indigestible carbohydrate which is metabolised to short chain **fatty acids** (SCFA) by microorganisms present in the human colon (b1) and which contains dietary fibre(s) and/or indigestible oligosaccharides. (A2) comprises an **oil** blend (a2), indigestible carbohydrate (b2) as above which is gum arabic, soy polysaccharide, hydrolysed inulin, fructooligosaccharide (FOS) or xylooligosaccharide (XOS). ; (c) nutrient(s) selected from beta -carotene, vitamin E, vitamin C, taurine and selenium; and (d) protein. (a1) contains at least 25 weight % of **oils** containing eicosapentenoic acid (20:5n3) (EPA) and **docosahexaenoic** acid (22:6n3) (DXE) and (A1) also contains (c). (a'1) comprises an **oil** blend containing the individual **fatty acids** oleic acid (18:1n9) 11.5-15.7%; **linoleic** acid (18:2n6) 6.6-9.0%; **alpha** - **linolenic** acid (18:3n3) 1.5-2.1%; EPA 15.1-20.5%; and DXE 6.3-8.6%. (a2) comprises 5-40% canola **oil**, 10-50% medium chain **triglycerides**, 25-80% **fish oil** 3-30% soybean **oil**, and 2-6% soy lecithin.
ADVANTAGE - The prod. is free from the side effects of drug therapy as used at present, using **antiinflammatory**, antibiotic, and/or immunosuppressive agents; and of total parenteral nutrition. The SCFA are beneficial; the carbohydrate (b) is metabolised to provide these without the need for rectal admin. The condition is also through to stem from **arachidonic** acid-derived **inflammatory** mediators; there is evidence that **n-3** acids inhibit this pathway to give an **antiinflammatory** effect, and also that the SCFA improve incorporation of **n-3** acids into colonocytes, reducing mucosal irritation. The components (c) are either antioxidants or are required for functioning of antioxidant enzymes to reduce oxygen radical mediated damage.
Dwg.0/5
FS CPI
FA AB; DCN
MC CPI: B03-A; B03-F; B03-H; **B04-B01B**; B04-B01C1; B04-B01C2; B04-C02D; B04-C02X; B04-N02; B05-A03B; B10-C02; B10-C04E; B14-C03;

B14-E08; B14-E10C; B14-E11; B14-S08

L142 ANSWER 10 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1995-122870 [16] WPIX
CR 1997-502374 [46]; 1998-347237 [30]
DNC C1995-056070
TI Enteral compsns. containing poly unsatd. fat and saponin - for treating or preventing infection or **inflammation.**
DC B05
IN CHAVALI, S; FORSE, R A; FORSE, R
PA (NEWE-N) NEW ENGLAND DEACONESS HOSPITAL
CYC 63
PI US 5397778 A 19950314 (199516)* 6 A61K031-38 <--
WO 9522971 A1 19950831 (199540) EN 15 A61K031-36 <--
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NL NO NZ PL PT RO RU SD SE SI SK TJ TT UA UZ VN
WO 9528163 A1 19951026 (199548) EN 17 A61K031-70 <--
RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW MX NL NO NZ PL PT RO RU SD SE SG SI SK TJ TT UA UZ VN
AU 9519282 A 19950911 (199550) A61K031-36 <--
AU 9522744 A 19951110 (199607) A61K031-70 <--
EP 804207 A1 19971105 (199749) EN A61K031-70 <--
R: BE CH DE FR GB IT LI NL
KR 97701043 A 19970317 (199813) A61K031-36 <--
JP 10500937 W 19980127 (199814) 17 A61K031-34 <--
JP 10504016 W 19980414 (199825) 23 A61K031-70 <--
EP 873116 A1 19981028 (199847) EN A61K031-36 <--
R: BE CH DE FR GB IT LI NL
ADT US 5397778 A **CIP of US 1994-201682 19940225, US 1994-228599 19940415**; WO 9522971 A1 **WO 1995-US2246 19950222**; WO 9528163 A1 **WO 1995-US3894 19950329**; AU 9519282 A **AU 1995-19282 19950222**; AU 9522744 A **AU 1995-22744 19950329**; EP 804207 A1 **EP 1995-916134 19950329, WO 1995-US3894 19950329**; KR 97701043 A **WO 1995-US2246 19950222, KR 1996-704747 19960826**; JP 10500937 W **JP 1995-522448 19950222, WO 1995-US2246 19950222**; JP 10504016 W **JP 1995-526982 19950329 , WO 1995-US3894 19950329**; EP 873116 A1 **EP 1995-911881 19950222, WO 1995-US2246 19950222**
FDT AU 9519282 A Based on WO 9522971; AU 9522744 A Based on WO 9528163; EP 804207 A1 Based on WO 9528163; KR 97701043 A Based on WO 9522971; JP 10500937 W Based on WO 9522971; JP 10504016 W Based on WO 9528163; EP 873116 A1 Based on WO 9522971
PRAI **US 1994-228599 19940415; US 1994-201682 19940225**
REP 12Jnl.Ref; DE 4017766; EP 387000; JP 2273622; JP 63157934; US 4752618; WO 8902275; DE 3001435; EP 183674; EP 253198; EP 305243; EP 534178; GB 2041942; WO 9523167
IC ICM A61K031-34; A61K031-36; A61K031-38; A61K031-70
ICS **A23L001-03; A23L001-30; A23L001-302; A23L001-304**; A61K031-23; A61K033-00; A61K035-60; A61K035-78
ICA C07D493-04
AB US 5397778 A UPAB: 19980730
Method for treating and preventing infection in persons at risk comprises enteral admin. of a formulation containing Quil A saponin (I) as its active ingredient, in conjunction with a source of dietary polyunsatd. fatty acids (II), provided that (II) constitute at least a significant part of the fat fed to the persons. Also claimed is an enteral formulation for treatment of infection or **inflammation** in a patient, having (I) as an active ingredient and comprising a source of (II), with the above

proviso.
The source of (II) is pref. a **fish oil** or vegetable **oil** rich in omega-3 fatty acids, especially a **fish oil** containing at least 10% omega-3 fatty acids. A lignan selected from sesamin, episesamin, sesaminol, episesaminol and sesamolin is pref. administered in conjunction with the enteral formulation
Dwg.0/0

FS CPI
FA AB; DCN
MC CPI: B04-A07E; **B04-B01B**; B10-C04E; B14-A01; B14-A02; B14-C03; B14-N

L142 ANSWER 11 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1992-433346 [52] WPIX
DNC C1992-192348
TI New emulsion for reducing blood tri **glyceride** and cholesterol levels - comprises vegetable and/or marine **oil**, an aqueous phase and a **phospholipid.**
DC B05 D13 E11 E17
IN LARSSON-BACKSTROEM, C; LARSSON-BACKSTROM, C; LARSSONBACKSTROEM, C
PA (KABI) KABI PHARMACIA AB; (FREP) FRESENIUS KABI AB; (PHAA) PHARMACIA & UPJOHN AB; (PHAA) PHARMACIA AB
CYC 25
PI

WO 9221335	A1	19921210	(199252)*	EN	32	A61K031-19	<--
RW: AT BE CH DE DK ES FR GB GR IT LU MC NL SE							
W: AU CA FI JP KR NO US							
AU 9219101	A	19930108	(199315)			A61K031-19	<--
PT 100524	A	19930831	(199338)			B01F003-08	<--
NZ 242697	A	19940225	(199411)			C08L091-00	<--
JP 06508123	W	19940914	(199441)			A61K031-20	<--
AU 657969	B	19950330	(199521)			A23D007-02	<--
EP 660708	A1	19950705	(199531)	EN		A61K031-19	<--
R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE							
US 5434183	A	19950718	(199534)		10	A61K031-66	<--
SG 64864	A1	20010116	(200109)			A61K031-66	
EP 660708	B1	20020417	(200227)	EN		A61K031-19	
R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE							
DE 69232564	E	20020523	(200241)			A61K031-19	
ES 2174833	T3	20021116	(200302)			A61K031-19	
JP 3387498	B2	20030317	(200323)		10	A61K031-20	

ADT WO 9221335 A1 **WO 1992-SE333 19920519**; AU 9219101 A **AU 1992-19101 19920519, WO 1992-SE333 19920519**; PT 100524 A **PT 1992-100524 19920527**; NZ 242697 A **NZ 1992-242697 19920512**; JP 06508123 W **JP 1992-511338 19920519, WO 1992-SE333 19920519**; AU 657969 B **AU 1992-19101 19920519**; EP 660708 A1 **EP 1992-911596 19920519, WO 1992-SE333 19920519**; US 5434183 A **WO 1992-SE333 19920519, US 1993-157024 19931229**; SG 64864 A1 **SG 1996-1678 19920519**; EP 660708 B1 **EP 1992-911596 19920519, WO 1992-SE333 19920519**; DE 69232564 E **DE 1992-632564 19920519, EP 1992-911596 19920519, WO 1992-SE333 19920519**; ES 2174833 T3 **EP 1992-911596 19920519**; JP 3387498 B2 **JP 1992-511338 19920519, WO 1992-SE333 19920519**
FDT AU 9219101 A Based on WO 9221335; JP 06508123 W Based on WO 9221335; AU 657969 B Previous Publ. AU 9219101, Based on WO 9221335; EP 660708 A1 Based on WO 9221335; US 5434183 A Based on WO 9221335; EP 660708 B1 Based on WO 9221335; DE 69232564 E Based on EP 660708, Based on WO 9221335; ES 2174833 T3 Based on EP 660708; JP 3387498 B2 Previous Publ. JP 06508123, Based on WO 9221335
PRAI **SE 1991-1642 19910530**
REP DE 3347269; DE 3721137; SE 8705122; US 4820731; WO 8600523; WO 8702247
IC ICM A23D007-02; A61K031-19; A61K031-20; A61K031-66; B01F003-08; C08L091-00

ICS A23D005-00; A23D007-00; A23D009-00; A23J007-00; **A23L001-30**; A61K009-10; A61K009-107; A61K009-127; A61K031-23; A61K037-22; A61P029-00; A61P037-06; B01F017-00; B01F017-14; B01F017-34; C07F009-10

AB WO 9221335 A UPAB: 19931118
In an emulsion containing vegetable **oil** and/or marine **oil**, an aqueous phase and **phospholipid** (I). (I) is of marine and/or synthetic origin and contains at least 30 weight % **omega-3 - fatty acids** (03FA).

Also new are (I) use of such (I) as therapeutic agents and (2) mono-, bi- and/or multi-layered vesicles containing such (I). Specifically, 03FA are DHA (**docosahexaenoic** acid) and EPA (eccosapentaenoic acid). The emulsion contains 0.5-40 weight volume % **oil** and 0.1-30 weight % volume % (I), opt. also one or more biologically active cpds.

USE/ADVANTAGE - The new nutritional emulsions (for infant or total parenteral nutrition) result in lower blood levels **triglycerides** and cholesterol than similar emulsions containing the same amount of 03FA as **fish oil.** (I) also have **antiinflammatory** and/or immunosuppressant effects (especially for treating rhematoid arthritis and **sepsis**) and affect normal brain and retinal development and function. Vesicles are useful as carriers for drugs or diagnostic reagents and opt. include a receptor-specific ligand to improve targetting. When admin. as (I), 03FA (and also **omega-6 fatty acids** present in the vegetable **oil** component) are incorporated at higher levels into biological membranes
Dwg.0/7

FS CPI
FA AB; DCN
MC CPI: **B04-B01B**; B04-B01C; B05-B01P; B10-C04E; **B12-A01**; B12-D02; B12-D02B; B12-D07; B12-H03; B12-M03; D03-C; D03-F07; **D03-H01T**; E05-G09C; E05-G09D

ABEQ US 5434183 A UPAB: 19950904
An emulsion comprising vegetable and/or marine **oil**, aq. phase and **phospholipids** as emulsifier contains omeqa fatty acids, viz, DHA and EPA, in amt. 30+ % wt.. Amt. **oil** is 0.5-40 % wt. and **phospholipids** 0.1-30 % wt.. Other bioactive components may be present. The form may be as mono-, bi- and/or multi-layered vesicles.

USE - Compsns. give low blood **triglyceride** and cholesterol levels and are used in treatment of **inflammatory** and immunosuppressive disorder e.g. rheumatoid arthritis and **sepsis** and also have effect on normal brain and retina development.
Dwg.0/7

L142 ANSWER 12 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1992-378172 [46] WPIX
DNC C1992-168000
TI Immuno-potentiating prod. containing **omega-3 fatty acid**, ester, glycerine, **phospholipid** - when administered in early post-operative recovery, increases NK cell activity preventing complications i.e. infection.
DC B05 D13
PA (NISS) NISSHIN FLOUR MILLING CO
CYC 1
PI JP 04279523 A 19921005 (199246)* 4 A61K031-23 <--
ADT JP 04279523 A **JP 1991-65717 19910111**
PRAI **JP 1991-65717 19910111**
IC ICM A61K031-23
ICS A23D009-00; A61K037-22
ICA **A23L001-30**
AB JP 04279523 A UPAB: 19931116
Product contains **omega-3 fatty acid** or ester, **phospholipid** or **glyceride** that have **omega-3 fatty acid** as **fatty**

acid components. In composed **fatty acid**, content as **omega-3 fatty acid** is regulated to 1-8 fold against **omega-6 fatty acid.**

USE/ADVANTAGE - Prod. increases activity of NK cell. By administration in an early stage of post operation, decrease of immunopotency can be prevented and complication (infection) can be prevented. NK cell shows phagocytosis against infused foreign body (especially virus) at once, different from T cell or B cell. It keeps complication (infection) at a minimum, until T cell differentiates, and antibody is produced from B cell. The fat processing product is per intestine preparation as NK cell activity increasing preparation It can be administered as capsules, liquids, granules, fine subtilise, powders, suspensions, emulsions, etc. It can be administered concomitantly in common per intestine nutrient. Daily dose for an adult is 0.1 - 50 g pref. 0.3 - 40 g as **omega-3 fatty acid.** It can also be used by adding with common nutritive food.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: **B04-B01B**; B10-C04E; B10-G02; **B12-A01**; **B12-A06**; B12-D02B; B12-J01; D03-C; **D03-H01T**

L142 ANSWER 13 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1990-194842 [26] WPIX

DNC C1990-084229

TI Dietetic compsn. or pharmaceutical containing blackcurrant **oil** - for preventing **inflammation**, platelet aggregation and metastatic cancer.

DC B04 D13

IN BUCHANAN, M; CROZIER-WILLI, G; FLEITH, M; CROZIERWIL, G

PA (NEST) SOC PROD NESTLE SA; (WILL-I) CROZIER-WILLI G; (NEST) NESTEC SA

CYC 20

PI	EP 374591	A	19900627 (199026)*				<--
	R: AT BE CH DE ES FR GB IT LI LU NL SE						
	PT 92690	A	19900629 (199031)				<--
	AU 8945547	A	19900628 (199034)				<--
	NO 8905183	A	19900625 (199034)				<--
	CA 2004332	A	19900623 (199036)				<--
	JP 02221228	A	19900904 (199041)				<--
	ZA 8909632	A	19900926 (199044)				<--
	CH 676909	A	19910328 (199116)				<--
	US 5141958	A	19920825 (199237)		7	A61K031-20	<--
	EP 374591	B1	19921104 (199245)	FR	17	A61K037-22	<--
	R: AT BE CH DE ES FR GB IT LI LU NL SE						
	DE 68903378	E	19921210 (199251)			A61K037-22	<--
	US 5234952	A	19930810 (199333)#		7	A61K031-20	<--
	ES 2045363	T3	19940116 (199407)			A61K037-22	<--
	IE 62603	B	19950222 (199519)			A61K031-19	<--
	CA 2004332	C	20000718 (200045)	EN		A61K035-00	
	JP 3284132	B2	20020520 (200236)		10	A61K035-78	

ADT EP 374591 A **EP 1989-122506 19891206**; JP 02221228 A **JP 1989-334614 19891222**; ZA 8909632 A **ZA 1989-9632 19891215**; US 5141958 A **US 1989-446130 19891205**; EP 374591 B1 **EP 1989-122506 19891206**; DE 68903378 E **DE 1989-603378 19891206**, **EP 1989-122506 19891206**; US 5234952 A **Div ex US 1989-446130 19891205**, **US 1992-890542 19920528**; ES 2045363 T3 **EP 1989-122506 19891206**; IE 62603 B **IE 1989-3878 19891205**; CA 2004332 C **CA 1989-2004332 19891130**; JP 3284132 B2 **JP 1989-334614 19891222**

FDT DE 68903378 E Based on EP 374591; US 5234952 A Div ex US 5141958; ES 2045363 T3 Based on EP 374591; JP 3284132 B2 Previous Publ. JP 02221228

PRAI **CH 1988-4790 19881223**

REP EP 92076; EP 92085; EP 902076
IC ICM A61K031-19; A61K031-20; A61K035-00; A61K035-78; A61K037-22
ICS **A23L001-21**; A61K031-215; A61K031-23; A61P009-00
AB EP 374591 A UPAB: 19930928
Use of blackcurrant **oil** to prepare a dietetic compsn. or pharmaceutical capable of improving the bioavailability of **dihomogamma linolenic** and **eicosapentaenoic** acid in relation to the bioavailability of **arachidonic** acid, is new.
USE/ADVANTAGE - The dietetic compsn. or pharmaceutical can be used to prevent (a) **inflammatory** conditions, (b) platelet aggregation and thromboses and (c) the proliferation and spreading of metastatic cancer. Dosage is 1-25g blackcurrant **oil**/day, especially 2-5g of blackcurrant pip **oil**/day.
0/0
FS CPI
FA AB
MC CPI: B04-B01C1; B12-D07; B12-G07; B12-H02; B12-J01; **D03-H01T**
ABEQ EP 374591 B UPAB: 19930928
The use of a blackcurrent **lipid** for the preparation of a dietetic or pharmaceutical composition for promoting the bioavailability of **dihomogammalinolenic** acid and **eicosapentaenoic** acid over the bioavailability of **arachidonic** acid.
0/0
ABEQ US 5141958 A UPAB: 19930928
A new treatment for cancer metastases comprises admin. **lipids** from blackcurrrant seed to selectively reduce inter cell adhesion. Dosage is 1-25 g/day of **lipids.** Pref. 2-5 g/day blackcurrant seed **oil** po., p.e. or rectally, opt. as dietetic compsn.
ADVANTAGE - EPA and DHLA are provided while bioavailability of AA is depressed, without the disadvantages of ingesting **fish oil**
0/0
ABEQ US 5234952 A UPAB: 19931119
Thrombosis prevention comprises admin. of **lipids** obtd. from blackcurrent seeds in an effective amt. to reduce thrombogenicity of blood vessels. The **lipid** is extracted blackcurrent seed **oil**, a mixt. of atty acids obtd. by the hydrolysis or fractionation of the **oil**, a pharmaceutically acceptable salt of the fatty acids, a mixt. of the fatty acids with glycerol or a mixt. of all of them. A fat-soluble antioxidant may be included e.g. tocopherols, ascorbic acid, etc..
USE/ADVANTAGE - Used for prevention of **inflammatory** diseases, platelet adhesion, platelet aggregation, thromboses, etc., for the proliferation and dissemination of cancerous metostases and for inhibiting adhesion of immune cells. The compsns. obtd. from the **lipid** promote the bioavailability of dihomagummalinolenic acid and **eicosapentaenoic** acid over **arachidonic** acid. Admin. is oral, enteral, reactal or parenteral.
Dosage is e.g. 1-25g **lipid**, pref. 2-5g **oil** per day in a single dose or pref. 2 or 3 doses.
Dwg.0/0

L142 ANSWER 14 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1989-099868 [13] WPIX
CR 1986-042032 [06]
DNC C1989-044116
TI New tri **glyceride**(s) and dietary supplements - containing omega-3 and medium-chain fatty acid components.
DC B05 C03 D13 E13
IN BABAYAN, V K; BISTRIAN, B R; BLACKBURN, G L; MASCIOLI, E A
PA (NEWE-N) NEW ENGLAND DEACONESS HOSPITAL
CYC 15

PI WO 8902275 A 19890323 (198913)* EN 25 <--
RW: AT BE CH DE FR GB IT LI LU NL SE
W: AU JP
AU 8825354 A 19890417 (198929) <--
US 4871768 A 19891003 (198949) 6 <--
EP 374188 A 19900627 (199026) <--
R: AT BE CH DE FR GB IT LI LU NL SE
JP 02502010 W 19900705 (199033) <--
EP 374188 B1 19931103 (199344) EN 10 A61K037-22 <--
R: AT BE CH DE FR GB IT LI LU NL SE
DE 3885462 G 19931209 (199350) A61K037-22 <--
CA 1324155 C 19931109 (199351) C11C003-08 <--
EP 374188 A4 19900926 (199513) <--
JP 2722229 B2 19980304 (199814) 6 C07C069-587 <--

ADT WO 8902275 A **WO 1988-US3037 19880831**; US 4871768 A **US 1987-92438 19870903**; EP 374188 A **EP 1988-908842 19880831**; JP 02502010 W **JP 1988-508081 19880831**; EP 374188 B1 **EP 1988-908842 19880831, WO 1988-US3037 19880831**; DE 3885462 G **DE 1988-3885462 19880831, EP 1988-908842 19880831, WO 1988-US3037 19880831**; CA 1324155 C **CA 1988-576566 19880906**; EP 374188 A4 EP 1988-908842 ; JP 2722229 B2 **JP 1988-508081 19880831, WO 1988-US3037 19880831**

FDT EP 374188 B1 Based on WO 8902275; DE 3885462 G Based on EP 374188, Based on WO 8902275; JP 2722229 B2 Previous Publ. JP 02502010, Based on WO 8902275

PRAI **US 1984-630732 19840712; US 1987-92438 19870903**

REP 2.Jnl.Ref; US 4272548; EP 120169

IC A23D009-00; **A23L001-30**; A61K031-22; A61K037-22; C07C069-58
ICM A61K037-22; C07C069-587; C11C003-08
ICS A23D009-00; A23D009-007; **A23L001-30**; A61K031-22; A61K031-23; A61K035-60; A61K038-00; C07C069-58; C11C003-10

AB WO 8902275 A UPAB: 19950412
New synthetic **triglycerides** (I) contain at least one omega-3 fatty acid gp. and at least one caprylic and/or capric acid gp. Also claimed are dietary supplements, (II) containing 10-40% of an 'oily **lipid** fraction' comprising 10-90 weight% medium-chain **triglycerides** and 10-50 weight% of omega-3 fatty acids.

(I) are of formula R1OCH2-CHOR2-CH2OR3, where R1 and R3 = caprulic and/or capric acid gps. and R2 = an omega-3 fatty acid gp. (I) are prepared by conventional transesterification, especially using a **fish oil** as omega-3 fatty acid source.

(II) may also contain 1-2 weight% lecithin, 1-3 weight% of an osmolarity modifier (especially glycerol) and 0-90% of an **oil** containing omega-9 fatty acids, especially a high-oleic-acid vegetable **oil.**

USE/ADVANTAGE - (I) and (II) are useful for animal and especially human nutrition. They promote resistance to infection and cardiovascular problems, esp,. in patients suffering from bacterial infections, surgical trauma, burns, malnutrition, ageing or cancer and those with secondary immunosuppression due to chemotherapy or diabetes.

Dwg.0/0

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B10-C02; B10-E04C; **B12-A01**; B12-D02A; B12-F01C; B12-J01; B12-L09; C10-C02; C10-E04C; **C12-A01**; C12-D02A; C12-F01C; C12-J01; C12-L09; D03-G01; **D03-H01T**

ABEQ US 4871768 A UPAB: 19930923
New synthetic **triglyceride** (I) has glycerol backbone with 3 fatty acids attached. Acids are selected from a 1st. gp. of omega3 fatty acids and a 2nd gp. of caprylic acid and/or capric. At least one fatty acid is from each of gp.s 1 and 2, pref. 1 from 2nd. these pref.being bound to non-adjacent C atoms, the whole comprising a rearranged

structured **lipid.** The omega3 acids may be from plant. plankton, fungal, or **fish oils** esp. menhaden, salmon, anchovy or herring **oil.**(New method of minimising the effects of infection and subsequent infection comprises admin. e.g. as supplement or as total p.e. nutrition, of 10-80 wt.% Oily fraction of (I) or 10-40 % wt. oily fraction having 10-90% of (I), and also 1-2% **phospholipid** emulsifier and 1-3% osmolarity modifier (glycerol).

ADVANTAGE - Good absorption of calories promotes resistance to , and mimimises effects of infection in vulnerable patients. (I) does not simulate insulin release or block reticiloednothelical system.

ABEQ EP 374188 B UPAB: 19931213
A synthetic **triglyceride** comprising a glycerol backbone having three fatty acids attached thereto, said fatty acids being selected from a first group consisting of omega 3 fatty acids, and a second group consisting of caprylic acid, and/or capric acid, whereby at least one of said fatty acids is selected from said first group and at least one (for example two) of said fatty acids in selected from said secvond group, and whereby if two of said fatty acids are selected from said second group, said two fatty acids are bound to adjacent carbon atoms of said glycerol backbone.
Dwg.0/0

L142 ANSWER 15 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
AN 1986-042032 [06] WPIX
CR 1989-099868 [13]
DNC C1986-017870
TI Minimising effects of infection - by administering a diet rich in **omega-3 fatty acids.**
DC B05 C03 D13
IN BABAYAN, V K; BISTRIAN, B R; BLACKBURN, G L; MASCIOLI, E
PA (MASC-I) MASCIOLI E A; (NEWE-N) NEW ENGLAND DEACONS
CYC 14
PI WO 8600523 A 19860130 (198606)* EN 23 <--
RW: AT BE CH DE FR GB IT LU NL SE
W: AU JP
AU 8546326 A 19860210 (198619) <--
EP 188573 A 19860730 (198631) EN <--
R: AT BE CH DE FR GB IT LI LU NL SE
JP 61502816 W 19861204 (198703) <--
US 4752618 A 19880621 (198827) <--
US 4820731 A 19890411 (198917) <--
AU 9063751 A 19910509 (199126) <--
EP 188573 B 19920318 (199212) 13 <--
R: AT BE CH DE FR GB IT LI LU NL SE
DE 3585681 G 19920423 (199218) <--
JP 07059508 B2 19950628 (199530) 7 A61K031-20 <--
ADT WO 8600523 A **WO 1985-US1332 19850711**; EP 188573 A **EP 1985-903749 19850711**; JP 61502816 W **JP 1985-503321 19850711** ; US 4752618 A **US 1984-630732 19840712**; US 4820731 A **US 1989-207122 19890615**; EP 188573 B **EP 1985-903749 19850711**; JP 07059508 B2 **JP 1985-503321 19850711**, **WO 1985-US1332 19850711**
FDT JP 07059508 B2 Based on JP 61502816, Based on WO 8600523
PRAI **US 1984-630732 19840712**; **US 1987-92438 19870903**; **US 1988-207122 19880615**
REP 4.Jnl.Ref; DE 3213744; SSR870520; EP 106571
IC A23D007-00; **A23L001-30**; A61K031-23; A61K035-60
ICM A61K031-20
ICS A23D007-00; **A23L001-30**; A61K031-23; A61K035-60
ICA **A23L001-29**
AB WO 8600523 A UPAB: 19950412
A method of minimising the effects of infection comprises administering a diet rich in **omega-3 fatty acids.**

These are pref. **oils** derived from herring, anchovy, cod and menhaden. A prefd. dietary supplement comprises 10-20 weight% oily fraction containing **omega-3 fatty acids**, 1-2 weight% of an emulsifier (e.g. egg yolks, **phospholipids** and soybean **phospholipids**), 1-3 weight% of an osmolality modifier (e.g. glycerin) and sterile water.

USE/ADVANTAGE - By replacing the predominantly **omega-6 fatty acid** containing **oils** with **omega-3 fatty acid**-containing **oils**, the levels of type 2 prostaglandins is reduced and the levels of type 3 prostaglandins is increased. Patients infected with wound infections, empyemas, bacteremias, abscesses and **septicemias**, may be treated. Patients at high risk of infection are also treated e.g. patient with secondary immunosuppression doe to chemotherapy or diabetes mellitus, protein-malnourished patients and patients undergoing abdominal surgery.

Dwg.0/0

Dwg.0/0

FS CPI

FA AB

MC CPI: **B04-B01B**; B04-B01C2; **B12-A01**; B12-A02C; **B12-A06**; B12-A07; B12-B04; B12-J01; **C04-B01B**; C04-B01C2; **C12-A01**; C12-A02C; **C12-A06**; C12-A07; C12-B04; C12-J01; **D03-H01T**

ABEQ DE 3585681 G UPAB: 19930922

A method of minimising the effects of infection comprises administering a diet rich in **omega-3 fatty acids.**

These are pref. **oils** derived from herring, anchovy, cod and menhaden. A prefd. dietary supplement comprises 10-20 wt.% oily fraction contg. **omega-3 fatty acids**, 1-2 wt.% of an emulsifier (e.g. egg yolks, **phospholipids** and soybean **phospholipids**), 1-3 wt.% of an osmolality modifier (e.g. glycerin) and sterile water.

USE/ADVANTAGE - By replacing the predominantly **omega-6 fatty acid** contg. **oils** with **omega-3 fatty acid**-contg. **oils**, the levels of type 2 prostaglandins is reduced and the levels of type 3 prostaglandins is increased. Patients infected with wound infections, empyemas, bacteremias, abscesses and **septicemias**, may be treated. Patients at high risk of infection are also treated e.g. patient with secondary immunosuppression doe to chemotherapy or diabetes mellitus, protein-malnourished patients and patients undergoing abdominal surgery.

ABEQ EP 188573 B UPAB: 19930922

The use of **omega3 fatty acids** for the manufacture of a dietary material for minimising the effects of infection in animals (e.g. humans) other than avians.

ABEQ US 4752618 A UPAB: 19930922

Infection and subsequent infection in high risk patients is minimised by administering a diet which is controlled in its caloric and **fatty acid** intake. Diet contains 10-20 wt.% of mixt. of **fatty acid**-contg. **oils** which comprise **omega 6 fatty acids**, and 10-90% of **omega 3 fatty acids** each or their prim **fatty acids.** Pref. **omega 3 oils** comprise **oils** derived from herring, anchovy or menhaden.

USE - In treatment of patients infected with wound infections, empyemae, bacteremias, abscesses, or **septicemias**, as a result of sec. immunosuppression due to chemotherapy or diabetes mellitus, protein-malnourishment or abdominal surgery.

ABEQ US 4820731 A UPAB: 19930922

Dietary supplement comprises higher unsatd. **fatty acid**

oils (at least 10 emulsifier (1-2 wt. %); glycerol (1-3 wt. %) as an osmolality modifier; and water. The oily component comprises a mixt. of **omega-3-fatty acid oils** (10-90 wt.%) and **omega-6-fatty acid oils** as the major components.

Pref. emulsifier are egg yolk **phospholipids** and soya bean **phospholipids.**

USE - The prods. are suitable for parenteral administration.

=> d his

(FILE 'HOME' ENTERED AT 09:35:24 ON 31 AUG 2004)
SET COST OFF

FILE 'HCAPLUS' ENTERED AT 09:35:33 ON 31 AUG 2004

L1	1	S	(WO2000-EP8731 OR EP99-118173)/AP,PRN
		E	NESTLE/PA,CS
L2	2617	S	NESTLE?/PA,CS
		E	TURINI M/AU
L3	25	S	E3-E6
		E	ROESSLE C/AU
L4	8	S	E3,E4
		E	ROSSLE C/AU
L5	7	S	E3,E4
		E	BREUILLE D/AU
L6	34	S	E3,E4
		E	CROZIER W/AU
L7	5	S	E3,E9,E10
		E	WILLI G/AU
		E	CROZIER G/AU
L8	24	S	E3-E8
		E	FINOT P/AU
L9	80	S	E4-E7
		E	RICHELLE M/AU
L10	45	S	E3,E4,E8
		E	DUTOT G/AU
L11	11	S	E3,E5

FILE 'REGISTRY' ENTERED AT 09:45:51 ON 31 AUG 2004

L12	6	S	(LINOLEIC ACID OR Γ-LINOLENIC ACID OR DIHOMO-Γ-LI
L13	9	S	(EICOSAPENTAENOIC ACID OR DOCOSAPENTAENOIC ACID OR DOCOSAHEXA
L14	1	S	92661-11-5
L15	7	S	L12,L14
L16	1	S	DIHOMO-Γ-LINOLEINIC ACID/CN
L17	7	S	L15,L16

FILE 'HCAPLUS' ENTERED AT 09:52:10 ON 31 AUG 2004

L18	52030	S	L17
L19	1471	S	L17(L) FFD/RL
L20	1280	S	L17(L) THU/RL
L21	20593	S	L18 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L22	63109	S	(ARACHIDONIC OR LINOLEIC OR GAMMA LINOLENIC OR DIHOMO GAMMA()
L23	24880	S	L22 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
		E	FATTY ACIDS/CT
		E	FATTY ACIDS (L) N/CT
		E	FATTY ACIDS (L) OMEGA/CT
		E	FATTY ACIDS (L) POLYUNSAT/CT
L24	2403	S	E13,E16
L25	2519	S	FATTY ACID?/CT (L) (N6 OR (N OR OMEGA)()6 OR OMEGA6)
L26	276	S	L24,L25 (L) FFD/RL
L27	168	S	L24,L25 (L) THU/RL
L28	2167	S	L24,L25 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?

```
L29       30232 S L19,L20,L21,L23,L26,L27,L28
L30       26049 S L13
L31        1830 S L13 (L) FFD/RL
L32        1316 S L13 (L) THU/RL
L33       14009 S L30 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L34       12812 S (EICOSAPENTAENOIC OR DOCOSAPENTAENOIC OR DOCOSAHEXAENOIC OR A
L35        8135 S L34 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
                E FATTY ACIDS (L) POLYUNSAT/CT
L36        5489 S E12,E14,E15
L37        5703 S FATTY ACIDS?/CT (L) (N3 OR (N OR OMEGA)()3 OR OMEGA3)
L38         855 S L36,L37 (L) FFD/RL
L39         654 S L36,L37 (L) THU/RL
L40        5131 S L36,L37 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?
L41       17717 S L31,L32,L33,L35,L38,L39,L40
L42       11526 S L29 AND L41
L43       21471 S L18-L29 AND L30-L41
L44        5431 S L43 AND LIPID#/CW
                E MEDIUM CHAIN TRIGLYCERIDE/CT
                E E11+ALL
L45        1538 S E2
L46        2489 S ((MED OR MEDIUM OR M)()CHAIN)(L)(GLYCERIDE OR TRIGLYCERIDE)
                E FISH OIL/CT
                E E5+ALL
L47        4257 S E2
L48        1570 S L47 (L) (FFD/RL OR THU/RL)
L49        3744 S L47 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR
L50         715 S L47-L49 AND LIPID#/CW
L51          85 S L47-L49 AND L45,L46
L52        5950 S L44,L50,L51
                E SEPSIS/CT
L53        7317 S E3,E5-E10
                E E8+ALL
L54        8136 S E1,E2,E3
                E E3+ALL
L55        3750 S E4+NT
                E E3+ALL
L56       10652 S E3+NT
                E SHOCK/CT
L57        4729 S E4-E12
                E E4+ALL
L58       13891 S E8,E9,E7+NT
                E E26+ALL
L59        6857 S E3,E2+NT
L60           8 S L52 AND L53-L59
L61         152 S L1-L11 AND L18-L60
L62          17 S L61 AND L47-L49
L63          55 S L61 AND L42,L43
L64          30 S L63 AND LIPID
L65          41 S L62,L64
L66          23 S L62,L63 NOT L65
L67           8 S L52 AND ?SEPSI?
L68          13 S L52 AND ?SEPTI?
L69          17 S L60,L67,L68
L70           1 S L61 AND L69
L71          14 S L69 AND (PY<=1999 OR PRY<=1999 OR AY<=1999)
L72           3 S L69 NOT L70,L71
L73           1 S L72 AND SEPTIC RAT
                SEL DN AN L71 2 6 12 13
L74          10 S L71 NOT E1-E12
L75          11 S L73,L74,L70
L76          11 S L75 AND (N6 OR N 6 OR OMEGA6 OR OMEGA 6 OR N3 OR N 3 OR OMEGA
L77           8 S L76 AND (LINOLEIC OR ?LINOLENIC? OR ARACHINDONIC OR DOCOSADIE
L78          11 S L75-L77 AND L1-L11,L18-L77
```

```
     FILE 'HCAPLUS' ENTERED AT 10:26:26 ON 31 AUG 2004

     FILE 'MEDLINE' ENTERED AT 10:37:40 ON 31 AUG 2004
L79        14314 S L17
L80        40760 S L22
L81         4507 S (N6 OR OMEGA6 OR (N OR OMEGA)()6)(L)FATTY ACID
L82        43258 S L79-L81
L83         3226 S L13
L84         6867 S L34
L85         7356 S (N3 OR OMEGA3 OR (N OR OMEGA)()3)(L)FATTY ACID
L86        11440 S L83-L85
L87         4872 S FISH OIL
                 E FISH OIL/CT
                 E E4+ALL
L88         9187 S E4+NT
                 E FATTY ACIDS/CT
L89         6700 S E88+NT
L90         8276 S E112+NT
L91        43258 S L82,L90
L92        10055 S L85,L89
L93         5250 S L91 AND L92
L94        10919 S L88,L93
                 E MEDIUM CHAIN/CT
                 E TRIGLYCERIDE/CT
                 E E5+ALL
                 E E3+ALL
L95         1344 S E3+NT AND L94
L96         4273 S LIPID AND L94
L97         4758 S L95,L96
                 E SEPSIS/CT
                 E E3+ALL
L98        52791 S E4+NT
                 E SHOCK/CT
                 E E3+ALL
L99        35591 S E5+NT
L100          17 S L97 AND L98,L99
L101          15 S L97 AND SHOCK
L102          28 S L97 AND (?SEPSIS? OR ?SEPTIC?)
L103          37 S L100-L102
L104          25 S L103 AND PY<=1999
                 SEL DN AN 1 5 9 10 11
L105          20 S L104 NOT E1-E10
L106          12 S L103 NOT L104
                 SEL DN AN 8 10
L107          10 S L106 NOT E11-E14
L108          30 S L105,L107 AND L79-L107
L109          30 S L108 AND (OMEGA? OR N6 OR N3 OR FATTY ACID OR ?GLYCERIDE? OR

     FILE 'MEDLINE' ENTERED AT 10:52:08 ON 31 AUG 2004

     FILE 'WPIX' ENTERED AT 10:52:28 ON 31 AUG 2004
L110           1 S L1
L111       27932 S LIPID/BIX OR (B04-B01B OR C04-B01B)/MC
L112         308 S L111 AND FISH OIL/BIX
L113          26 S L111 AND ((N3 OR OMEGA3)(L)FATTY ACID)/BIX
L114         305 S L111 AND (((N OR OMEGA)()3)(L)FATTY ACID)/BIX
L115         510 S L111 AND (EICOSAPENTAEN? OR DOCOSAPENTAEN? OR DOCOSAHEXAEN? O
L116          10 S L111 AND ((N6 OR OMEGA6)(L)FATTY ACID)/BIX
L117         140 S L111 AND (((N OR OMEGA)()6)(L)FATTY ACID)/BIX
L118         890 S L111 AND (LINOLEIC OR GAMMA LINOLENIC OR DIHOMOGAMMALINOLENIC
L119         344 S L113,L114,L115 AND L116,L117,L118
L120         601 S L112,L119
```

```
L121          11 S L120 AND (SEPSIS OR SEPTICEM? OR SEPTICAEM?)/BIX
L122          18 S L120 AND (B14-S06 OR C14-S06 OR B12-A01 OR C12-A01 OR B12-A06
L123         104 S L120 AND (P220 OR P420)/M0,M1,M2,M3,M4,M5,M6
L124         118 S L120 AND ?INFLAM?/BIX
L125           6 S L120 AND SHOCK/BIX
L126          39 S L121-L125 AND D03-H01T?/MC
L127          36 S L121-L125 AND A23L001/IPC
L128          53 S L126,L127
L129          19 S L128 AND PY<=1999
L130          28 S L128 AND PRY<=1999
L131          27 S L128 AND AY<=1999
L132          28 S L129-L131
L133          25 S L128 NOT L132
L134          22 S L132 AND (SHOCK OR ?INFLAM?)/BIX
L135           8 S L132 AND L121,L122
L136          26 S L134,L135
                 SEL DN AN 2 3 4 5 7 8 11 15 16 22 25
L137          15 S L136 NOT E15-E37
L138          15 S L110,L137
L139          13 S L138 AND ?LIPID?/BIX
L140           8 S L138 AND ?GLYCERID?/BIX
L141           9 S L138 AND OIL/BIX
L142          15 S L138-L141

     FILE 'WPIX' ENTERED AT 11:22:07 ON 31 AUG 2004

=>
```